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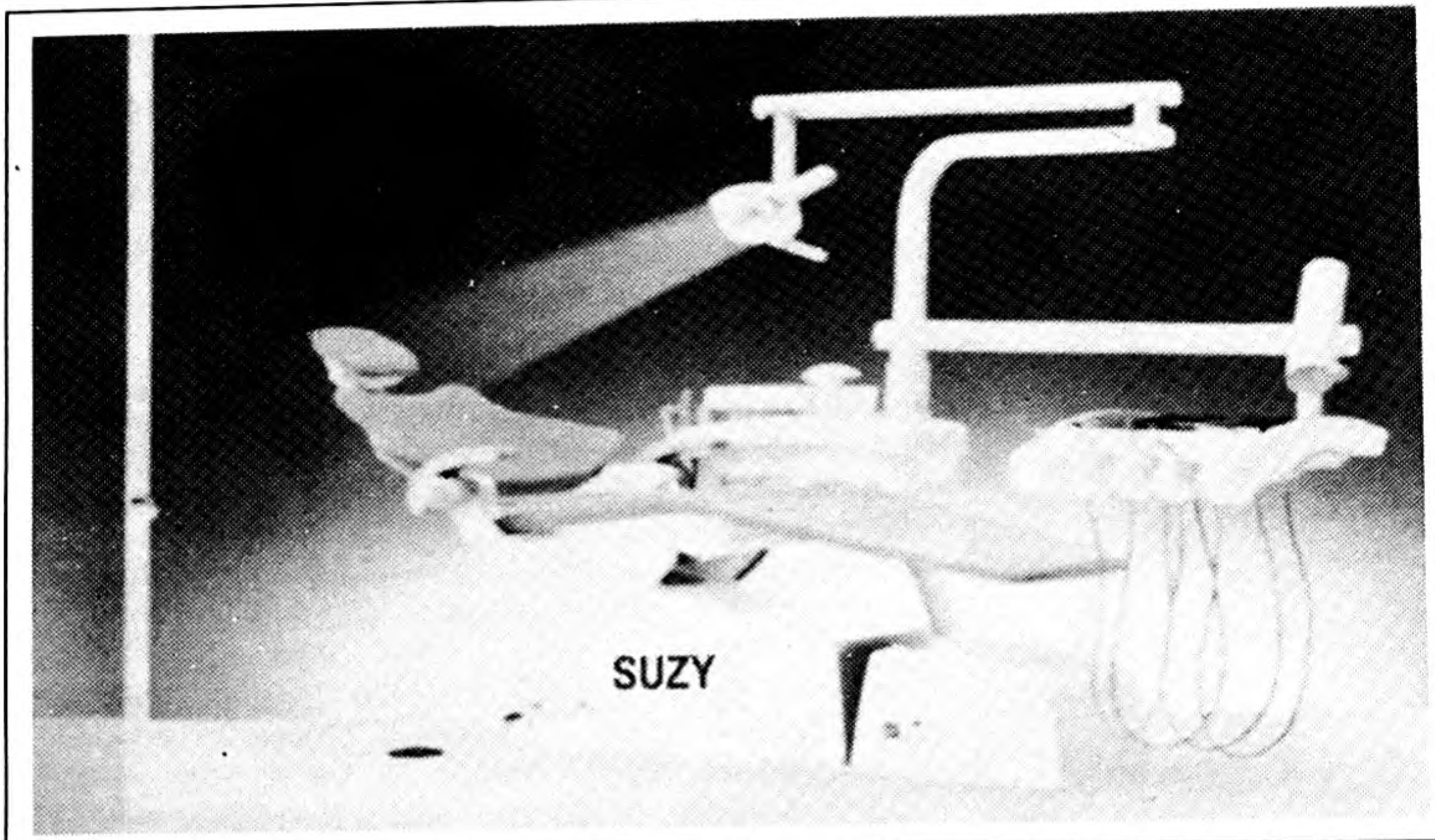

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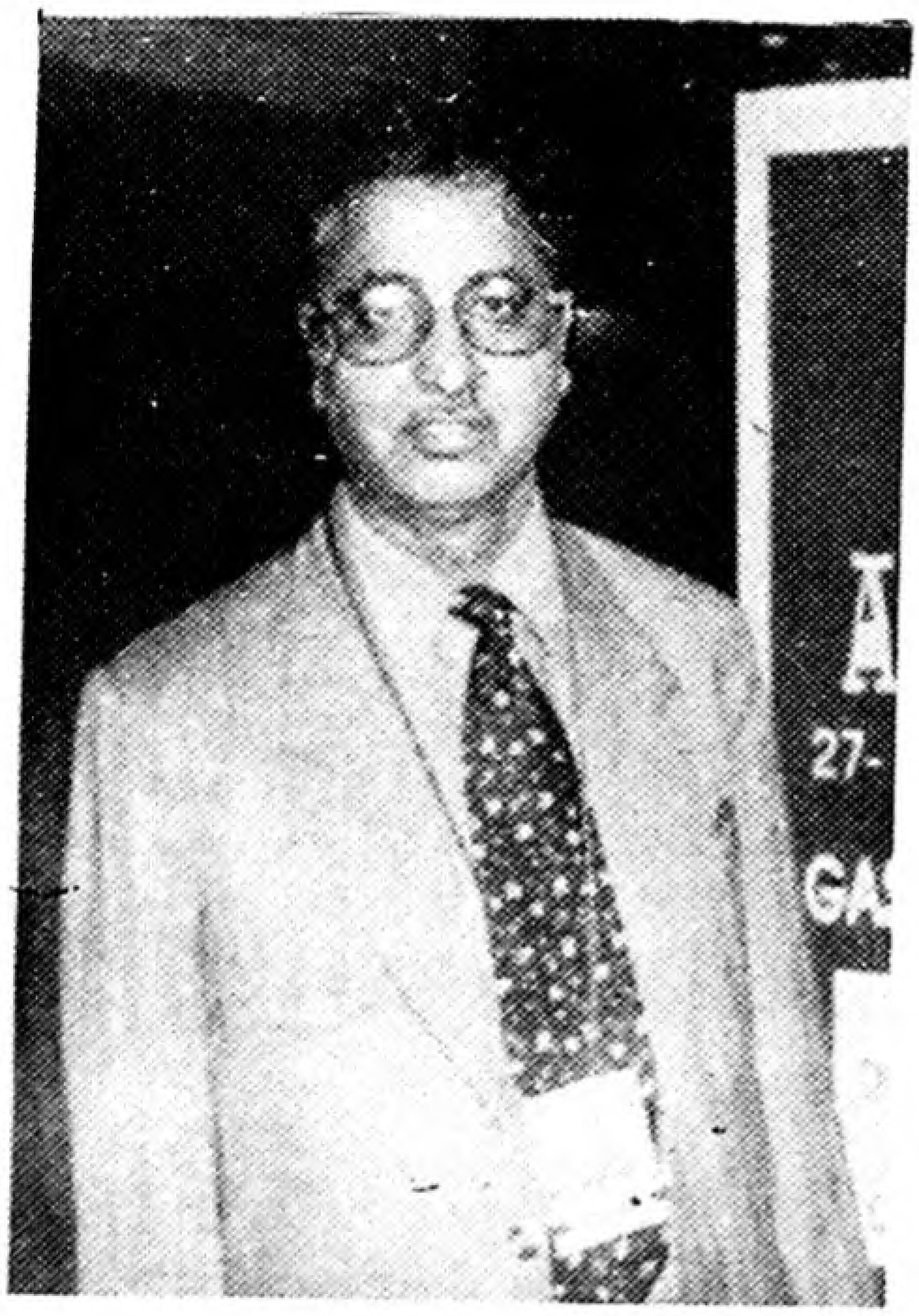

It gives me immense sense of satisfaction to present before you this issue. This is a special occasion for us, not because it is a new year issue bit because it marks our entry into tenth year of publication. Although the journal may have physically changed over the years but the core remains the same-to present high quality international research with special reference to this part of the globe.

A decade is a short time to make a palpable presence in the highly competitive market of medical publishing. There are small but significant pointers that we have not done badly .E mails asking for permission to use our articles for their clients coming from reputed international concerns prove a point. Demand for specimen copies from them are increasing. It was astonishing for some of our readers when a query in the "PUBMED" search engine produced JK-Pract citation on the very first page-download.Our journal is being cross referenced internationally,more often.

Year 2002 brought happy news.JK-Practitioner was accepted for inclusion in IndMED-an extensive database of about 75 Indian biomedical Journals. The decision by the experts was taken after thoroughly reviewing the journal for a couple of years. We have signed a memorandum of understanding with the National Informatics Center (Ministry of Information Technology,Govt. of India).The journal will be available online (Full text articles) on their website http://indmed.nic.in This publication is already indexed in EMBASE/ EXCERPTA MEDICA and listed in the Ulrich's Periodicals Directory (USA) and INSDOC (India).

A special meeting (The First Decade) is scheduled for July this year-a time for an international conference at Srinagar, when most of our editors from abroad will be gathered here. A future course of the journal will be under consideration.

I am thankful to the overseas consulting editors ,who have been a real help in shaping the journal to international standard .My team here, under the able guidance of the ever efficient executive editor, Dr.Zaffar Abbas are doing a remarkable job under difficult circumstances. My thanks go to the advertisers who are helping a "cause".Their contributions (Specialized publishing is expensive) go a long way in easing some of our problems.I acknowledge their help.

Wishing you a happy new year.

Prof. G.M. Malik M.D., F.A.C.G.
Srinagar

EOSINOPHILIC GASTROENTERITIS

Rafiq A. Sheikh, MB, MD, MRCP(UK), FACP, FACG; Thomas Prindiville, MD.; Shagufta Yasmeen, MD; MD, MRCP; Boris H. Ruebner, MD.

Eosinophilic gastroenteritis (EG) is an uncommon inflammatory disease, first described by Kaijser in two patients with syphilis who were allergic to neoarsphenamine[1]. Since then a little less than 300 cases have been reported in the literature[2-5]. The exact etiology is not known although most patients have a history of food allergies or intolerance and many have a family history of allergic disorders. Almost all patients have tissue eosinophilia, most have peripheral eosinophilia and raised IgE levels. Majority of the cases have favorable response to steroids suggesting a type-1 hypersensitivity reaction. The basic defect in EG may be an alteration in the mucosal integrity of the gut resulting in localization of various antigens in the gut wall and inducing tissue and blood eosinophilia. Specific food antigens can cause mast cell degranulation in the gastrointestinal wall, releasing eosinophil chemotactic factors and platelet activating factors. The degranulation of eosinophils causes release of histamine, cationic proteins like major basic protein (MBP), eosinophil peroxidase (EPO) and cytokines like tumor necrosis factor-à (TNF-à). These proteins promote inflammation, tissue damage and further mast cell degranulation resulting in a vicious circle. Degranulation of activated eosinophils and release of the cationic protein has been demonstrated in the small bowel of patients with EG and MBP and EPO have been found to be toxic to intestinal epithelial cells in guinea pigs[6-7]. Some cases of EG are associated with unrecognized parasitic infestations and allergic or toxic reaction to drugs. An outbreak of eosinophilic entero-colitis due to canine hookworm, Ankylostoma caninum was reported from Queensland, Australia[8-9]. Drugs like gold, azathioprine, carbamazepine, clofazimine and co-trimoxazole have been reported to cause eosinophilia with variable involvement of the gastrointestinal tract[10-11]. Eosinophilic gastroenteritis may involve the whole gastrointestinal tract. The clinical presentation (Table 1) may vary depending on the level and extent of involvement of the different layers of the digestive tract. Klein and Talley et al classified eosinophilic gastroenteritis into mucosal, submucosal and subserosal disease on the basis of predominant involvement[2,7,12]. Patients with predominant mucosal disease usually present with nausea, voniting, diarrhea nad weight loss. Patients with predominant muscular involvemenyt present with intermittent obstructive symptoms and sometimes complication like perforation or aspiration. Eosinophilic ascites is the hallmark of subserosal disease usually associated with intense peripheral eosinophilia. Rarely, eosinophilic gastroenteritis may involve the pleura, pericardium, bladder, pancreas, gall bladder, liver and the biliary tree.

The diagnosis is established by demonstrating eosinophilic infiltration on biopsies obtained on endoscopy, laparoscopy or laparotomy. An enzyme-linked immunosorbent assay has been developed in Australia to diagnose Ankylostoma canis but has not been evaluated outside of Australia. Barium studies, CT scanning and ultrasonography may all reveal thickening of the mucosal folds with or without nodular filling defects or gastric outlet obstruction depending on the severity and the site of eosinophilic infiltration. The CT scan may also demonstrate ascites, pleural effusions and lymphadenopathy in some cases[2-4]. The radiological differential diagnosis includes lymphoma, carcinoma, hypersecretory disease, Menetrier's disease, Whipple's disease, amyloidosis and intestinal lymphangiectasia. The endoscopic findings may be patchy and may vary from normal mucosa to mild erythema, thickened mucosal folds, nodularity and frank ulceration[2,4]. Full thickness surgical biopsies may be required if the disease process is confined to the muscle layer.

Inflammatory Fibroid Polyps are localized, non-neoplastic, reactive submucosal polyps or nodules of unknown etiology first described by Helwig and Ranier[13]. They involve the gastric antrum in 70% of cases and small bowel in 20 % and may cause pyloric obstruction and small bowel intussusception. It may be difficult to differentiate IFP from eosinophilic gastroenteritis (Table 2). Some investigators believe they may represent an intermediary stage in the evolution of eosinophilic gastroenteritis[14-18].

In a review of 220 cases Naylor reported a history of allergy in 52% cases[3], Lee et al in 4 out of 8 patients[4] and Lee et al reported it in 4 out of 5 cases[5]. Food allergy has been reported in 50% cases[2,12,19]. In a review of 220 cases

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stomach was the most frequently involved organ (43 % of cases). Duodenum and rest of the small bowel are also frequently involved[2,3]. Although EG can involve the whole gastrointestinal tract, the esophagus and colon are uncommonly involved[2,20]. Liver, biliary tract and gall bladder involvement is rare[2-4].

Table 1. Classification and clinical features of eosinophilic gastroenteritis

A. Mucosal and submucosal disease (most common)	
History of allergic disease	50%
Food intolerance/ allergy	50%
Abdominal Pain	77-100%
Nausea and vomiting	48- 62%
Diarrhea	42- 62%
Malabsorption	unusual
Protein losing enteropathy	unusual
Bleeding	rare
Intestinal perforation	rare
B. Muscle layer disease (may be localized)	
	Food intolerance/ allergy uncommon
	Crampy abdominal pain
	Nausea and vomiting
	Pyloric/ Intestinal obstruction
C. Serosal/ Subserosal disease (least common)	
	Allergic history common
	High eosinophil count
	Bloating
	Ascites
	Pleural effusion (rare)

Compiled from references Lee, Lee, Naylor

Table 2. Comparison of Eosinophilic Gastroenteritis and Inflammatory Fibroid polyps

Features	Eosinophilic Gastroenteritis	Inflammatory fibroid polyp
Age of onset	4th - 5th decade	6th -7th decade
Male: Female ratio	2 : 1	1.3 : 1
History of Allergy	~ 50%	Rare or Absent
Peripheral Eosinophilia	~ 80%	Absent
Tissue Eosinophilia	Always present	Present (variable)
Site of involvement	Stomach/ Small bowel	Antrum, Small bowel
Gross pattern	Diffuse, Multiple	Localized, solitary

Compiled from ref, Johnstone and Morson

The peripheral eosinophil count was normal in both these cases. Peripheral eosinophilia has been reported in up to 80% of cases[2]. Patients with predominantly serosal disease have the higher absolute eosinophil counts (average 8, 000/ L) than patients with mucosal disease (average 2,000/ dL) and muscle layer disease (average 1,000/ dL)[3]. Iron deficiency anemia and/ or evidence of malabsorption has been seen in 20-30% patients, especially those with mucosal disease[2]. IgE levels are more likely to be high in children with eosinophilic gastroenteritis than adults[21]. IFP is the most important differential diagnosis. Typically the polyps in FIP are submucosal in origin and involve the gastric antrum in 70% cases and small bowel in 20 %. They may cause pyloric obstruction and small bowel intussusception depending on the site of involvement. Histologically the lesions are characterized by a concentric stroma of blood vessels, fibroblasts, histiocytes, lymphocytes, polymorphonuclear leukocytes with variable eosinophilic infiltration[22]. Most of the patients require endoscopic resection of the polyps and some require surgical excision if the polyps are large[23]. Obstructive lesions are seen in patients with predominant muscular disease. However, patients with mixed serosal and mucosal disease may have a mixed clinical presentation. Patients with predominant muscular disease are less likely to have history of allergies and peripheral eosinophilia. Althogh benign lesions like peptic ulcer disease accounted for most of the cases of gastric out let obstruction in the past, with the evolution of effective therapy for peptic ulcer disease, malignancy has emerged to be the most important etiology[24]. It is, therefore, imperative to rule out malignancy in these patients. Other uncommon causes of gastric outlet obstruction include Crohn;s disease, post-surgical strictures, pancreatic pseudocyst, gall stones and chronic pancreatitis. Gastric outlet obstruction/ small bowel obstruction is an unusual presentation of eosinophilic gastroenteritis. In children this may mimic congenital pyloric stenosis[25]. There have been only few case reports of jejunal and ileal strictures and stenosis[26,27]. Eosinophilic gastroenteritis can cause edema, fibrosis and distorsion around the ampulla and even cause pancreatitis[28].

Steroids are the mainstay of treatment in eosinophilic gastroenteritis and about 90% patients respond to this therapy[3,29]. Patients with serosal disease usually show a dramatic response to steroids[3]. Azathioprine may be helpful as a steroid sparing agent in patients requiring high doses for maintenance. Sodium cromoglycate is a mast cell stabilizer and prevents release of toxic mediators like histamine, platelet activating factors and leukotrienes from mast cells. There have been reports of beneficial response to this drug[2,30-31]. The usual dose is 200 mg tid or qid. Ketotifen is similar to sodium cromoglycate in its biological profile and may be useful in some cases[32]. Elimination of presumed dietary articles is unhelpful in most cases[2]. Generally, eosinophilic gastroenteritis has a good prognosis. Fatalities from eosinophilic gastroenteritis are rare and usually due to perforation of the gut[33-34].

References

1. Kaijser R. Zur Kenntnis der allergischen Affektioner desima verdanungskannal von standpunkt desima chirugen aus. Arch Klin Chir 1937; 188: 36-64.
2. Talley NJ, Shorter RG, Philips SF, et al. Eosinophilic Gastroenteritis: a clinicopathological study of patients with disease of the mucosa, muscular layer, and subserosal tissues.

Gut 1990; 31 54-8.

3. Naylor AR. Eosinophilic Gastroenteritis. Scott Med J 1990; 35: 163-5.
4. Lee CM, Changchien CS, Chen PC, et al. Eosinophilic Gastroenteritis: 10 year experience. Am J Gastroenterol 1993: 88: 70-4.
5. Lee M, Hodges WG, Huggins TL, et al. Eosinophilic Gastroenteritis. Southern Med J 1996; 89: 189-94.
6. Tropier G, Colombel J F, Mathieu-Chandelier C, et al. Eosinophilic gastroenteritis: Ultrastructural evidence for a selective release of eosinophilic major basic protein. Clin Exp Immunol 1988; 74: 404.
7. Talley NJ, Kephart GM, McGovern TW, et al. Depsosition of eosinophil granule major basic protein in eosninophilic gastroenteritis and celiac disease. Gastroenterology 1992; 103; 137-45.
8. Walker NI, Croese J, Clouston AD, et al. Eossinophilic enteritis in Norteastern Australia. Pathology, association with Ancylostma caninum and implications. Am J Surg Pathol 1995; 19: 328-37.
9. Croese J, Loukas A, Opdebeeck J, et al. Occult enteric infection by Ancylostoma caninum: A previously unrecognized zoonosis. Gastroenterology1994; 106: 3
10. Michet CJ Jr, Rakela J, Luthra HS. Auranofin associated colitis and eosinophilia. Mayo Clin Proc 1987; 62: 142.
11. Anttila VJ, Valtonen M. Carbamazepine-induced eosinophilic colitis. Epilepsia1992; 33: 119.
12. Klein NC, Hargrove RL, Sleisenger MH, et al. Eosinophilic Gastroenteritis. Medicine 1970; 49: 299-319.
13. Helwig EB, Ranier A. Inflammatory Fibroid polyps of the stomach. Surg Gynecol Obstet 1953; 96: 355-67.
14. Tai YG, Liu JD, Lin KY, et al. Eosinophilic gastroenteritis with eosinophilic ascites. J Formosan Med Assoc. 1990; 89: 901-4.
15. Koch W, Stockmeyer E, Apablaza H. Eosinophilic granuloma of the stomach. Report of a case. AJR 1958; 80: 51-6.
16. Salmon PR, Pauley JW. Eosinophilic granuloma of the gastrointestinal tract. Gut 1967; 8: 814.
17. Suen KC, Burton JD. The spectrum of eosinophilic infiltration of the gastrointestinal tract and its relationship to other disorders of angiitis and granulomatosis. Hum Pathol 1979; 10: 31-43.
18. Ureles AL, Alschibaja T, Lodico D, et al. Idiopathic eosinophilic infiltration of the gastrointestinal tract, diffuse and circumscribed. Am J Med 1961; 30: 899-909.
19. Scudamore HH, Phillips SF, Swedlund HA, et al. Food allergy manifested by eosinophilia, elevated immunoglobulin E level and protein losing enteropathy: the syndrome of allergic gastroenteropathy. J Aller Clin Immunol 1982; &0: 129.
20. Mitsunobu M, Kiyoshi H, Yukari M, et al. Eosinophilic gastroenteritis involving the entire digestive tract. Am J Gastroenterol 1995; 90: 1868-70.
21. Katz AJ, Goldman H, Grand RJ. Gastric mucosal biopsy in eosinophilic (allergic) gastroenteritis. Gastroenterology 1977; 73: 705.
22. Johnstone JM, Moroson BC. Inflammatory fibroid polyp of the gastrointestinal tract. Histopathology 1978; 2: 349-61.
23. Hizawa K, Iida M, Tada S, et al. Endoscopic evaluation of gastric inflammatory fibroid polyp. Surgical Endoscopy 1995; 9: 397-400.
24. Cello JP. Eosinophilic gastroenteritis: a complex disease entity. Am J Med 1979; 67: 1097-1104.
25. Chowdhury A, Dhali GK, Banerjee PK. Etiology of gastric outlet obstruction. Am J Gastroenterol 1996; 91: 1679.
26. Hummer-Ehret BH; Rohrschneider WK; Oleszczuk-Raschke K; Darge K; Nutzenadel W; Troger J. Eosinophilic gastroenteritis mimicking idiopathic hypertrophic pyloric stenosis. Pediatric Radiology, 1998 Sep, 28(9):711-3.
27. Karande T; Oak SN; Trivedi A; Karmarkar S; Kulkarni B; Kalgutkar A. Proximal jejunal obstruction due to eosinophilic gastroenteritis. Journal of Postgraduate Medicine, 1996 Oct-Dec, 42(4):121-3.
28. Wig JD; Goenka MK; Bhasin DK; Vaiphei K. Eosinophilic gastroenteritis presenting as acute intestinal obstruction. Indian Journal of Gastroenterology, 1995 Jul, 14(3):104-5.
29. Maeshima A; Murakami H; Sadakata H; Saitoh T; Matsushima T; Tamura J; Karasawa M; Naruse T. Eosinophilic gastroenteritis presenting with acute pancreatitis. Journal of Medicine, 1997, 28(3-4):265-72.
30. Di Gioaccchino M, Pizzicannella G, Fini N, et al. Sodium cromoglycate in the treatment of eosinophilic gastroenteritis. Allergy 1990; 45: 161-66.
31. Perez-Millan A, Martin-Lorente JL, Lopez-Morante A, et al. Subserosal eosinophilic gastroenteritis treated efficaciously with sodium cromoglycate. Dig Dis Sci 1997; 42: 342-44.
32. Melamed I, Feanny SJ, Sherman PM, et al. Benefit of Ketotifen inpatients with eosinophilic gastroenteritis. Am J Med 1991; 90: 310-14.
33. Felt-Bersma RJF, Meuwissen SGM, Van Velzen D. Perforation of the small intestine due to eosinophilic gastroenteritis. Am J Gastroenterol 1984; 79: 442.
34. Martin A, Castagliuolo I, Floreani A, De Lazzari F, et al. Eosinophilic gastroenteritis: Report of two atypical cases. Ital J Gastroenterol 1991; 23:81-5.

"MORTALITY AMONG ELDERLY PATIENTS WITH FRACTURE OF THE HIP"

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ABSTRACT: *The main purpose of this retrospective study was to evaluate one- year mortality rate among patients with proximal femoral fractures treated surgically and assess the influence of the patient's medical status and ASA grade on this parameter.*

212 patients above 65 years with a mean of 82.7 (range 65-100), consisting of 104 (49.1%) intertrochanteric fractures and 108 (51.9%) femoral neck fractures.

Sliding/compression screw was used in 104 (49.1%), hemiarthroplasty in 81(38.2%), cannulated screw fixation in 18 (8.5%) and total hip replacement in 9 (4.2%)

One- year mortality rate relative to age, sex, ASA grade, type of fracture, time of operation and type of operation.

Patients were identified and clinical data obtained through the Hospital's computerised record and case notes. Patient's status at one year was ascertained from the hospital records or Family Physician.

One- year mortality rate for the whole group was 28.8%. There was an exponential increase in mortality rate with an increase in ASA grade. The relationship between ASA grade and one- year mortality rate for the whole group ($P<0.001$), younger age group of 65- 84 years ($P<0.001$), older age group of 85-100 ($P=0.002$), females ($P=0.000$), intertrochanteric fractures ($P=0.007$), femoral neck fractures ($P=0.022$), early operation group of <2days ($P=0.000$) and sliding/compression screw ($P=0.007$) was statistically highly significant.

Statistically significant mortality is neither dependent on age, nor the type of fracture, nor the time and type of surgery per se, but is essentially a reflection of ASA status.

We confirm that ASA grading is a highly significant predictive determinant and final arbiter of surgical risk and mortality in hip fractures.

Key words: *Femoral Neck Fractures, Hip, Elderly, Mortality, ASA grade significance*

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INTRODUCTION

There have been conflicting reports in literature regarding mortality rate associated with the proximal femoral fractures. White et al[1] reported an increased one-year mortality when operative delay exceeded 24 hours and also found that ASA grading scale is a reliable index of surgical risk and predictor of mortality. Similarly, Hamlet et al[2] observed that ASA classification is a good predictor of mortality and surgery within 24 hours had a significantly lower mortality rate regardless of ASA grade.

The United Kingdom Audit Commission Report of 1995[3] states that patients with hip fractures, who are medically fit to undergo surgery, should be operated as soon as possible, citing the Royal College of Physicians' recommendation that such operations should be carried out within 24 hours. Beringer et al[4] reported that medically fit elderly patients have improved chance of survival when operated within 24 hours of admission. Sexson and Lehner[5] reported an increased one year mortality rate in relatively healthy patients who did not have an operation for more than 24 hours after admission, though in patients with at least three pre-existing medical conditions the delay was related to a reduction in mortality rate.

Zuckerman et al[6] could detect no significant difference in mortality among patients operated on the day of admission and those operated on the first or second day, but surgical delay of more than two calendar days following admission proved to be an adverse influence and an important predictor of one year mortality for elderly patients with hip fracture. Therefore they advocated surgery within two days following admission.

In contrast, Kenzora et al[7] reported that patients who underwent surgery within a day of admission had significantly higher one- year mortality rate than those operated between second and fifth day. They found that this relationship was even stronger in relatively healthy patients.

Parker and Pryor[8] in a study of 765 patients found no association between mortality and time of surgery, but noted a small increase in the incidence of pressure sores and

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pneumonia with delay in surgery. Davis et al[9] also concluded that the timing of operative treatment is not an important determinant of three- month mortality.

Table 1: Classification of Groups

The American Society of Anaesthesiologists (ASA)[11]

Grade I = normal, healthy patient
Grade II = patient with mild systemic disease
Grade III = patient with severe but not incapacitating systemic disease
Grade IV = patient with a severe incapacitating systemic condition which is a constant threat to life
Grade V = patient who is near to death.

***Time of surgery**

Group 1 - patients operated within 2 calendar days of admission
Group 2 - patients operated after 2 calendar days

Age groups

Group A 65-74 years

Group B 75-84 years

Group C 85-94 years

Group D 95-100 years.

*(*The time of operation was taken as the interval in calendar days between admission and operation, e.g. a patient admitted to hospital on a Monday and operated on Wednesday was considered to have an interval of two calendar days. This corresponds to the protocol adopted by Zuckerman et al[6]).*

Table 2a - Co-morbidities of patients

Medical Condition	No of patients
Cerebro-vascular accident	50
Dementia	50
Congestive cardiac failure	31
Hypertension	31
Chronic obstructive airway disease	30
Ischaemic heart disease	23
Cardiac arrhythmia	20
Cancer	20
Diabetes	16
Parkinson's disease	15
Renal disease	4
Others	137
Total	**427**

Table 2b - Mortality by the number of co-morbidities

No. of co-morbidities	No.of patients	Died (mortality %)
0	21	4 (19%)
1	43	11 (25.6%)
2	73	22 (30.1%)
3	45	14 (31.1%)
4	21	7 (33.3%)
5/>5	9	3 (33.3%)
Total	***212***	***61 (28.8%)***

AIMs of the Study

The main focus of the study was to determine the one year mortality rate of proximal femoral fractures treated surgically and any possible association between the age, sex, type of fracture, time of operation, type of operation, ASA grading and mortality.

Table 2c - Mortality by the day of operation

Day of operation	No. of patients	Died (mortality%)
0	11	3 (27.3%)
1	78	23(29.5%)
2	55	18(32.7%)
3	40	9 (22.5%)
4	12	3 (25%)
5/>5	16	5 (31.3%)
Total	212	61(28.8%)

Table 3 - One year mortality rate by the type of operation and sex

Operation and sex	No. of patients	Died	Mortality %
1. Sliding/compression Screw			
Male	24	10	41.7%
Female	80	23	28.8%
Total	104	33	32.1%
P= 0.172			
2. Hemiarthoplasty			
Male	25	8	32.0%
Female	56	13	23.2%
Total	81	21	25.9%
P=0.284			
3. Cannulated Screws			
Male	4	2	50%
Female	14	5	35.71%
Total	18	7	38.88%
P=0.515			
4. Total Hip Replacement			
Male	1	0	0%
Female	8	0	0%
Total	9	0	0%
P= Not applicable			

Grouped Data Definitions

ASA grades
I and II
Low ASA grades
III and IV
High ASA grades
Age ranges
65 to 74 and 75 to 84
Younger patients
85 to 94 and 95 to
101 Older patients

Question I

Significance of ASA grading in relation to mortality by grades

Table 4: Mortality by ASA grade: all patients

	ASA I	ASA II	ASA III	ASA IV	Total
Died	2	10	37	12	61
Survived	31	53	58	9	151
Total	33	63	95	21	212
(mortality %)	(6.1%)	15.9%)	(38.5%)	(57.1%)	(28.8%)

P < 0.001

Study Methods

The patients included in the study were aged 65 years and over, admitted with proximal femoral fractures to Hereford General Hospital between January 1 –Decemberr 31, 1995. Patients were identified through the hospital's computerised patient administration system and records in Orthopaedic Department, which serve as a source of information regarding age, sex, type of fracture, patient's medical condition, ASA grade, time of operation, type of operation and post-operative complications during hospitalisation.

The patients were classified into groups according to ASA grade, time of operation and age, as shown in table 1. Follow-up information on patients' status at one year was obtained either through the hospital records or Family Physician. Outcome at one year after fracture was measured in terms of mortality.

STATISTICAL ANALYSIS

The calculations were performed in Epi Table (part of EpiInfo the disease surveillance database program written by the Centre for Disease Control and Prevention) and SPSS.

Where there were more than two columns of data, the proportion [Died to Survived] was compared using the contingency table method of calculating chi squared and degree of freedom and hence the probability of the data being a chance occurrence.

The method computes an " expected value" for the number of surviving and deceased patients. However the method is not recommended if more than 10% of "expected value" are <5 or any "expected value" is <1. Unfortunately this occurred owing to a small patient numbers in some categories and had to be overcome by combining the data into larger groupings (see 'Grouped Data Definitions). This frequently resulted in a 2 x 2 format. Whenever this was the case, the Fisher Exact method was used which has greater flexibility as long as the row and column totals are greater than zero.

Question II

ASA grading in relation to mortality by age

Table 5: Mortality versus ASA grade grouping: younger patients

	Low ASA grades	High ASA grades	Total
Died	7	27	34
Survived	52	38	90
Total	59	65	124
(mortality %)	(11.9%)	(41.5%)	(27.4%)

Fisher exact test: One-tail P < 0.001

Question III

Significance of ASA grading in relation to sex and grades

Table 6: Mortality versus ASA grade grouping: older patients

	Low ASA grades	High ASA grades	Total
Died	5	22	27
Survived	32	29	61
Total	37	51	88
(mortality %)	(13.5%)	(43.1%)	(30.7%)

Fisher exact test: One-tail P=0.002

Table No7: Mortality versus ASA grade groupings: males

	Low ASA grades	High ASA grades	Total
Died	1	19	20
Survived	8	26	34
Total	9	45	54
(Mortality%)	(11.1%)	(42.2%)	(37%)

P= 0.078

Table No8: Mortality versus ASA grade groupings: females

	Low ASA grades	High ASA grades	Total
Died	11	30	41
Survived	76	41	117
Total	87	71	158
(Mortality %)	(12.6%)	(42.3%)	(25.9%)

P= 0.000

Question IV

Significance of ASA grading in relation to type of fracture and grades

Table No9: Mortality versus ASA grade groupings: intertrochanteric fractures

	Low ASA grades	High ASA grades	Total
Died	5	28	33
Survived	29	42	71
Total	34	79	104
(Mortality%)	(14.7%)	(40%)	(31.7%)

P= 0.007

STUDY RESULTS

212 patients were included in the study, consisting of 158 (74.5%) females and 54 (25.5%) males. Their ages

Table No10: Mortality versus ASA grade groupings: femoral neck fractures

	Low ASA grades	High ASA grades	Total
Died	6	22	28
Survived	36	44	80
Total (Mortality%)	42 (14.3%)	66 (33.3%)	108 (25.9%)

P= 0.022

Question V

ASA grading in relation to the time of operation

Table11: Mortality versus ASA grade groupings: early operation (<2 days)

	Low ASA grades	High ASA grades	Total
Died	7	37	44
Survived	57	43	100
Total	64	80	144
(Mortality %)	(10.9 %)	(46.3 %)	(30.6 %)

P=0.000

Table 12: Mortality versus ASA grade groupings: late operation (>2 days)

	Low ASA grades	High ASA grades	Total
Died	5	12	17
Survived	27	24	51
Total	32	36	68
(Mortality %)	(15.6 %)	(33.3 %)	(25 %)

P=0.079

Question VI

Significance of ASA grading in relation to mortality by operation and grades

N.B. Since there were no deaths following total hip replacement, this procedure has not been included in Tables 17 18 and19.

Table 13:Mortality versus procedure (including THR): all ASA grades

	Sliding/ compression screw	Hemi-arthro-plasty	Cannulated screws	THR	Total
Died	33	21	7	0	61
Survived	71	60	11	9	151
Total	104	81	18	9	212
(mortality %)	(31.7%)	(25.9%)	(38.9%)	(0%)	(28.8%)

P=0.151

Table 14: Mortality versus procedure (excluding THR): all ASA grades

	Richards's screw	Hemi-arthro-screw	Cannulated screws	Total
Died	33	21	7	61
Survived	71	60	11	142
Total	104	81	18	203
(mortality %)	(31.7%)	(25.9%)	(38.9%)	(30.0%)

P=0.481

Table 15: Mortality versus procedure (excluding THR): low ASA grades

	Sliding/ compression screw	Hemi-arthroplasty	Cannulated screws	Total
Died	5	3	3	11
Survived	29	19	9	57
Total	34	22	12	68
(mortality %)	(14.7%)	(13.6%)	(33.3%)	(16.2%)

P=0.654

ranged from 65 to 100 years with a mean of 82.7. Majority of patients, 175(82.5%), were aged between 75 and 94 years.

104 (49.1%) patients sustained an intertrochanteric fracture and 108 (51.9%) a femoral neck fracture. 3 patients had fractures of both hips at different times during the year of study. Sliding/compression screw was used in 104 (45%), hemiarthroplasty in 81 (38.2%) cannulated screws in 18 (8.5%) and total hip replacement in 9 (4.2%). Sliding/compressions screw and hemiarthroplasty accounted for the bulk (87.2%) of operations.

The number of co-morbidities numbered 427, ranging from 0 to 5 per patient with a mean of 2. Major risk factors comprised cardiopulmonary compromise 84 (19.7%), cerebrovascular accident 65 (15.2%), dementia 50 (11.7%), hypertension 31(7.3%), malignancy 20 (4.7%) and diabetes 16 (3.7%).

The ASA grading of the patients was as follows- Grade I (33, 15.6%), Grade II (63, 29.7%), Grade III (95, 44.8%), Grade IV (21, 9.9%) and Grade V (0).

The timing of operation ranged from the day of admission to 11 days following admission with a mean of 2.2 days (standard deviation 1.78) and a median of 2 days.

19 (9%) patients developed post-operative complications whilst in hospital-urinary tract infection 5, revision procedures 3, wound infection 3, pulmonary embolism 2, deep venous thrombosis 2 and pressure sore 1.

One- year mortality was 28.8% for the whole group.

The deceased patients at one year after fracture were distributed through all age groups with 5 (17.9.%) out of 28 in the 65-74 years group, 29 (30.2%) out of 96 in the 75-84 years group, 27 (34.1%) out of 79 in the 85-94 years group, and 0 (0%) out of 9 in the 95-100 years group.

The outcome was also assessed by the type of operation. There were no deaths in the nine patients who had primary total hip replacement (THR). These patients included only one male. Their ages ranged from 69-85 years (mean 76 years). Operations were carried out between 1 and 3 days

Table16: Mortality versus procedure (excluding THR): high ASA grades

	Sliding/ compression screw	Hemi-arthroplasty	Cannulated screws	Total
Died	28	18	4	50
Survived	42	41	2	85
Total	12	59	6	135
(mortality %)	(40.0%)	(30.5%)	(66.7%)	(37.0%)

P=0.165

Table 17: Mortality versus ASA grade grouping: sliding/ compression screw

	Low ASA grades	High ASA grades	Total
Died	5	28	33
Survived	29	42	71
Total	34	70	104
(mortality %)	(14.7)	(40.0)	(31.7)

Fisher exact test: P=0.007

Table 18: Mortality versus ASA grade grouping: hemiarthroplasty

	Low ASA grades	High ASA grades	Total
Died	3	18	21
Survived	19	41	60
Tota			
(mortality %)	22 (13.6%)	59 (30.5%)	81 (25.9%)

Fisher exact test:P=0.102

Table 19: Mortality versus ASA grade grouping: cannulated screws

	Low ASA grades	High ASA grades	Total
Died	3	4	7
Survived	9	2	11
Total	12	6	18
(mortality%)	(25%)	(66.7%)	(38.9%)

Fisher exact test: P=0.117

following admission with a mean of 2.3 days. Their ASA status was I-II with the exception of one grade III. All were operated during daytime under consultant supervision. Mortality was also low in patients with lowest ASA grades (Grade I and II) who underwent sliding/ compression screw fixation (P=0.007). The type of operation otherwise had no predictive influence on mortality.

There was an exponential increase in mortality rate with increase in ASA grade: I-2 out of 33 (6.1%), II- 10 out of 63 (15.9%), III- 37 out of 95 (38.9%) and IV-12 out of 21 (57.1%).

Both in younger and older age groups, high ASA grades resulted in trebled mortality rate ($P<0.001$ and 0.002 respectively).

It was obvious that the relationship between ASA grade and one-year mortality rate for the whole group ($P<0.001$), younger age group of 65-84 years ($P<0.001$), older age group of 85-100 ($P=0.002$), females ($P=0.000$), intertrochanteric fractures ($P=0.007$), femoral neck fractures ($P=0.022$), early operation group of < 2days ($P=0.000$) and sliding/compression screw ($P=0.007$) was statistically highly significant.

DISCUSSION

The central focus of this study was to assess one-year mortality following proximal femoral fractures. The reported mortality in different series is subject to variations and conflicting conclusions.

Unlike Kenzora et al[7] we found that operative delay was not associated with statistically significant decrease in mortality rate

Zuckerman et al[10] in their prospective study found two calendar days is the watershed in the shift of mortality rate. As operative delay beyond two calendar days entailed a doubled risk of one-year mortality, they advocated surgery within two days. We adopted the same protocol in the division of our patients and found no statistical difference in mortality

Like White et al[1], our results confirm that the ASA grading scale is a reliable index of surgical risk and predictor of mortality. However, unlike White et al[1], who also concluded that mortality rate is inversely related to age, our study proved that mortality relative to ASA grading both in younger and older age groups alike is highly statistically significant. Hamlet et al[2] drew the same conclusion that ASA classification is a good predictor of mortality. They also concluded that surgery within 24 hours has significantly lower mortality regardless of preoperative ASA grading.

In our series, ASA grading proved to have highly significant statistical bearing on the group as a whole, both older and younger age groups, females, both intertrochanteric and femoral neck fractures, early surgical group and sliding/compression screw fixation. This extensive dominant influence of ASA grade is the predictive determinant of surgical risk and final arbiter of mortality in hip fractures.

Statistically significant mortality is neither dependent on the age, nor the type of fracture, nor the time and type of surgery per se, but is essentially a function of preoperative ASA grade.

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REFERENCES

1. White, B. L., Fisher, W. D. and Laurin, C. A. "Rate of mortality for elderly patients after fracture of the hip in the 1980s", *The Journal of Bone and Joint Surgery*, 1987; 69A(9): 1335-1340.
2. Hamlet, W. P., Lieberman, J. R., Freedman, E. L., Dorey, F. J., Fletcher, A., and Johnson, E. E. "Influence of health status and the timing of surgery on the mortality in hip fracture patients". *American Journal of Orthopaedics*, 1997;26: 621-627.
3. Audit Commission. "United they stand - co-ordinating care of elderly patients with hip fracture". *HMSO*, 1995.
4. Beringer, T. R., Crawford, V. L., and Brown, J. G. "Audit of surgical delay in relationship to outcome after proximal femoral fracture".*Ulster Medical Journal, 1996; 65: 32-38.*
5. Sexson, S. B. and Lehner, J. T. "Factors affecting hip fracture mortality". *Journal of Orthopaedic Trauma*, 1987;1, 298-305.
6. Zuckerman, J. D., Skovron, M. L., Koval, K. J., Aharonoff, G. and Frankel, V. H. "Postoperative complications and mortality associated with operative delay in older patients who have a fracture of the hip",*The Journal of Bone and Joint Surgery*, 1995; 77A(10): 1551-1556.
7. Kenzora, J. E., McCarthy, R. E., Lowell, J. D. and Sledge, C. B.. Hip fracture mortality, relation to age, treatment, pre-operative illness, time of surgery and complications". *Clinical Orthopaedics*. 1984;186: 45-56.
8. Parker, M.J. and Pryor, G.A. "Initial management". In *Hip Fracture Management*. Blackwell Scientific Publications, 1993; pp 67-68.
9. Davis, T. R.C., Sher, J. L., Porter, B. B. and Checketts, R. G."The timing of surgery for intertrochanteric femoral fractures". *Injury*. 1988;19; 244-246.
10. Zuckerman, J. D., Skovron, M. L., Keval, K. J., Aharonoff, G. and Frankel V. H. Correspondence to the editor. *Journal of Bone and Joint Surgery*, 1997; 79A: 470.
11. Owens, W. D., Felts, J. A. and Spitznagel, E. L. Jr. "ASA physical status classifications: a study of consistency of ratings" *Anaesthesiology*, 1978; 49; 239-243.

TREATMENT OF HALLUX VALGUS BY GIBSON AND PIGGOTT OSTEOTOMY

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ABSTRACT: *This study comprised of 50 patients of Hallux Valgus treated by Gibson and Piggott Osteotomy. There were 40 males and 8 females with an average age of 25 years. There were 84% good and 16% satisfactory results (Criteria of Gibson and Piggott 1962). No patient had poor result. Adequate lateral displacement of head was achieved in all cases so was prevention of dorsal angulation. Both these factors are essential for good results. Centralization of EHL tendon was done in all cases. Meticulous attention was paid to the application of plaster of paris cast in all cases. Postoperative Metatarsalgia was not a problem. This study emphasizes the role of simple procedure for a complex problem.*

Key Words: *Halus Valgus, Osteotomy.*

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INTRODUCTION:

Hallux Valgus has been defined by Stamm[1] (1957) as a complex progressive deformity affecting the forefoot in which a lateral deviation of great toe is the most obvious feature. The complex may involve rotation of Hallux, Metatarsus Primus varus, over riding of second toe, over riding of lateral toes and bunnionette of first metatarsal

Helal et al[2] (1974) found that in Britain 2000 operations are done in adolescents alone. There are 164 Operation described for Hallux Valgus (Stanley 1975), reflecting the inadequacy of these procedures in achieving results.

We describe the results of Gibson and Piggott Osteotomy for this condition, which we are doing routinely in our hospital with gratifying results.

METERIAL AND METHODS:

This study was conducted on 50 patients with hallux valgus from March 1992 to April1997. There were 40 females and 10 males in the study group. The age of patients ranged from 18-40 years with an average of 25 years. The longest period of follow up was five years with an average of three years. The indications of surgery were painful bursa over medial eminence of first metatarsal head, difficulty in obtaining a comfortable shoe and for cosmetic reasons.

Through a longitudinal dorsimedial incision, neck of the first metatarsal bone was exposed subperiosteally and divided with an Osteotome just proximal to joint capsule. The cortex of proximal fragment was trimmed so as to leave a stout lateral spike about 3/8th of an inch long near the planter than dorsal aspect. The metatarsal head was then impacted onto the spike with toe in neutral position. Making small hole in the distal fragment facilitated this. The metatarsal head was thus displaced laterally and little towards sole. Neither lateral tight structures were released nor any thing was done to the bunnion. However centralization of EHL tendon was made routinely. Wound was closed and plaster of Paris cast applied from great toe to above ankle in boot fashion. Patients were discharged from the hospital next day and advised crutch walking non-weight bearing. At six weeks plaster was removed and full weight bearing was allowed. No definite postoperative programming was prescribed

Application of Plaster of paris:

Meticulous attention must be paid to this step. After applying cotton from Nail fold to above ankle three slabs are prepared. One is applied on medial side of great toe to mid foot; another dorsal aspect of great toe and third one is applied around the great toe. Big toe is kept in neutral position and cast is completed up to ankle in a boot fashion.

Assessment of results

The patient's concerns before and after surgery were most important factors. They were pain over medial side of first metatarsal, pain under 2nd.3rd 4th metatarsal heads, problems with shoe ware, and appearance of foot. Postoperative factors were correction of deformity, preservation of mobility of the joint and reduction of width of forefoot (Gibson and Piggott[2] 1962). The results were classified as

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Good:

i. Completely free of symptoms.
ii. Deformity corrected as judged by surgeon and patient
iii. Normal mobility of joint

Satisfactory:

i. Symptom absent or reduced so as to cause no inconvenience
ii. Deformity considered by patient to be corrected.

Poor:

i. If any post-operative symptoms were trouble some
ii. Or patient considered operating valueless.

Bad:

If Patient thought foot was worst after surgery

Results:

We had 84% good and 16% satisfactory results. The reason for not having poor or bad result was that our patients were young and there was minimal subluxation of articular surfaces.

Table – 1

Result	Number	Percentage
Good	42	84
Satisfactory	8	16

Complications:

Many complications can arise during and after surgery as has been reported by many authors.

1. **Partial displacement of metatarsal head:** Radiographs taken during Ist Week after surgery showed incorrect angulation in 5 feet, which was corrected by manipulation under anesthesia. This we agree with Gibson et al that this may occur during application of POP. It did not affect final results and we suggest that meticulous attention should be paid while applying plaster of paris cast.
2. **Shortening of 1st metatarsal:** About 5mm of shortening does occur with this procedure on an average. We had 8mm of shortening in 9 patients. But none of our patients complained of shortening. Even making them aware they did not show much concern. Fear of shortening should not prevent surgeon from fashioning an adequate spike, which is needed for secure fixation.
3. **Nonunion:** Gibson and Piggot[2] (1962) reported this in 1 patient, which was treated by bone grafting. We did not encounter this type of complication.
4. **Metatarsalgia:** Hilal et al[3] (1974) observed that lengthening metatarsal at its base gives poor results by increasing pain and by reducing the movements at MCP joint. Kotharai[4] (1977) has also not been able to correlate the short first metatarsal with Metatarsalgia. We too are of the opinion that short metatarsal does not contribute to or increase Metatarsalgia.Gibson and Piggot[3] (1962) observed transfer metatarsalgia in 31 patients, which increased after surgery. Kothari[4] had similar observations in 24% patients. we did not observe this kind of complication in our series. Only 4 of our patients had pain under metatarsal but it did not bother them. According Mitchell et al[5] (1958) and agreed by Gibson and Piggott[2] (1962), dorsal angulation is an important cause of bad results. Dorsal angulation caused by displacement from normal weight bearing position of first metatarsal puts excessive strain on heads of lesser metatarsal especially 2nd and may produce Metatarsalgia. We analysed lateral X-rays of about 25 patients including 4 who complained pain in the forefoot. There was no evidence of dorsal angulation. One way of preventing dorsal angulation is by delayed weight bearing, which we adopted in all cases
5. **Inadequate lateral displacement of distal fragment:** Lateral displacement by which narrowing of forefoot is produced is essential feature of surgery. Meticulous attention must be paid to this step. This is the key to success in this type of surgery. We did not allow less than maximum displacement of head. This we consider is the reason why we did not have un-satisfactory results, which has been reported by Gibson and Piggott 1962.

Discussion:

We shall briefly describe the rule of peg in hole Osteotomy of metatarsal neck in the treatment of Hallux Valgus as devised by Gibson and Piggott (1962). 100% of adults in whom we performed this procedure gave good or satisfactory results. No other procedure for this condition has produced similar results.

Helal et al[3] (1974) investigated the results of several operations for this condition *(See table below)*. From this table it is evident that Wilson Osteotomy gave excellent or good results, which comes nearer to this procedure. In Wilson Osteotomy distal fragment is small and difficult to control. Wilson[6] has suggested that an angle of Osteotomy less than 45 degree produces instability and greater than 45 degrees is difficult to displace. Khotarri S.S[1] (1974) while reviewing Wilson's cases found that even 45-degree angulation is unstable and produces dorsal angulation. Wilson[6] (1963) does not recommend Osteotomy for middle-aged patients without giving reasons. In contrast Gibson and Piggott Osteotomy is transverse thus stable and easy to displace.

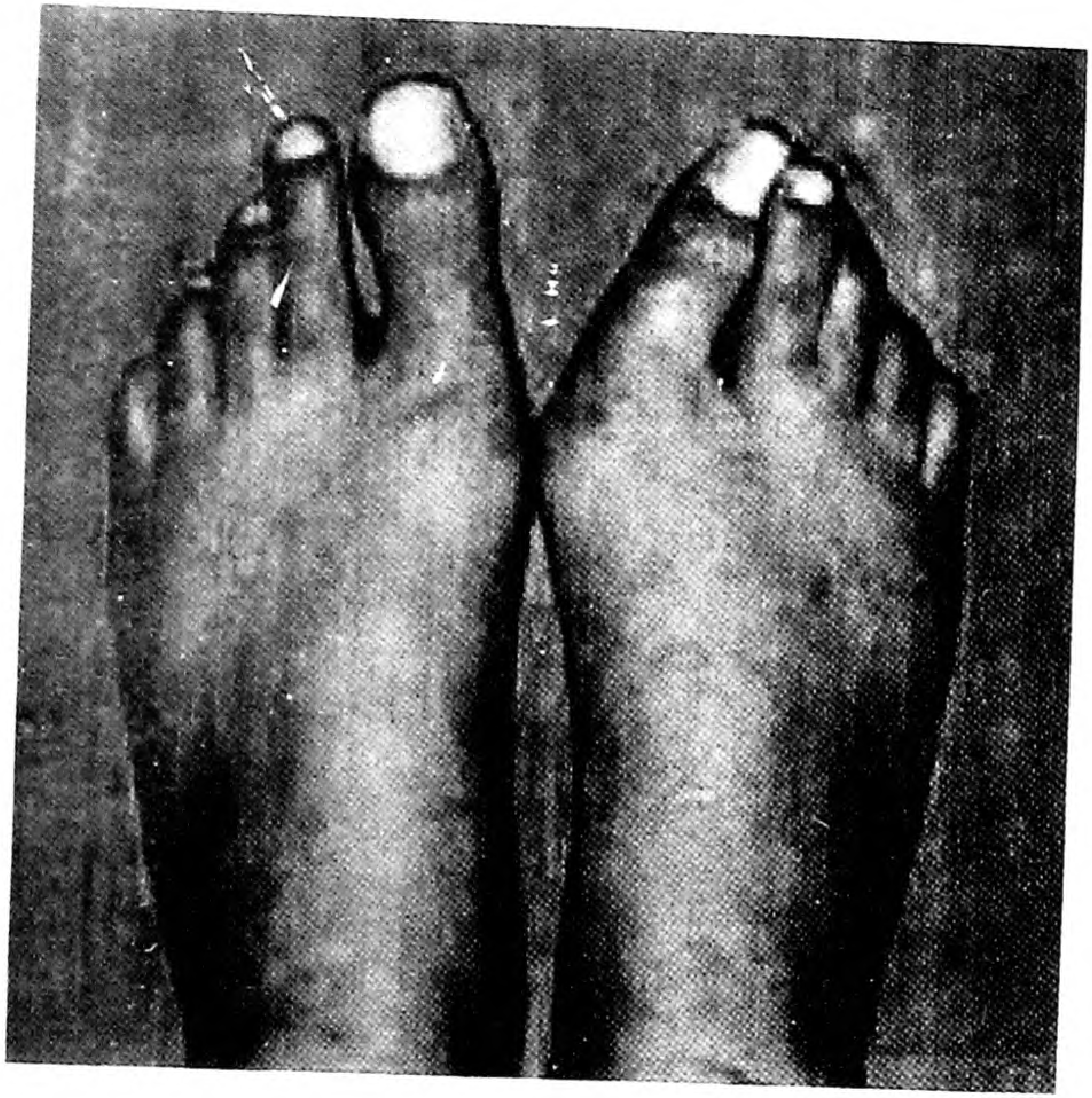

Fig. 1: Photograph showing Hallux valgus of right foot

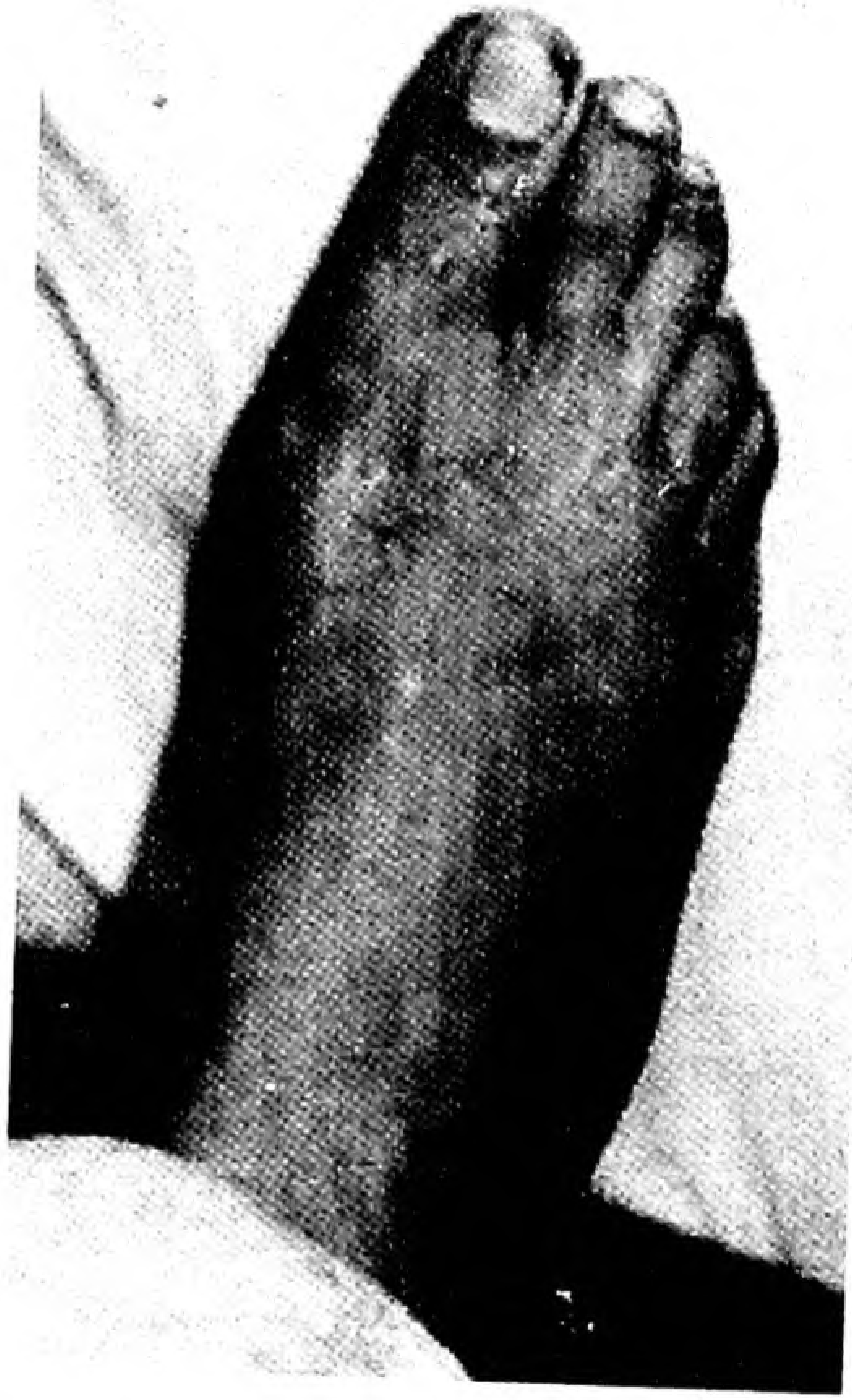

Fig. 2: Photograph showing correction after Gibbson and Piggott osteotomy

Table No.2

Operation	Excellent/ Good %	Poor
Simmonds	80	20
Golden	86	20
McBride Modified	46	54
Wilson	94	06
Peabody	66	34
Mittchell	80	20
Mac Bride	46	54

After Hilal et al[3] (1974)

Selection of Cases:

As per Gibson and Piggott there are two main contra-indications to Surgery; degenerative arthritis and Metatarsalgia. We had no Patient who had severe arthritis, as our series included young patients. However four patients had metatarsalgia before surgery but it did not get aggravated postoperatively. As has been already discussed, our observation regarding metatarsalgia does not correlate with Gibson and Piggott. We are also not in agreement with Kothari who had 24% of Metatarsalgia in his series. Whether this occurs in more patients in our series, perhaps another review after few years may throw more light on this.

This Osteotomy is valuable in a foot, which has moderate deformity; no arthritis and symptoms are entirely due to increased width of forefoot. This type of foot is commonly seen in adolescent and adults.

The principle effect of this surgery is to narrow forefoot without deranging metatarsophalangeal joint. In addition to this, lateral shift and proximal displacement of distal fragment relaxes the conjoined tendon thus permitting realignment of great toe (Gibson and Piggott) thus making capsular release unnecessary. We did not release any tissue or capsule .One constant finding in our cases was lateralisation of EHL tendon, which was acting more of a lateral force than a dorsiflexor. We routinely made centralization of EHL by bringing it to center by releasing lateral attachments, which we consider were abnormal. We did not do anything with bunnion.

As already discussed utmost attention must be paid to the operative technique. Lateral displacement is essential and to achieve this Osteotomy should be made as far distal in the broadest part of neck. It is most important to avoid dorsal angulation of metatarsal head thus the spike on proximal fragment must be towards planter aspect and great toe must be immobilized in slight planter flexion.

CONCLUSION:

Correction of Hallux Valgus by Gibson and Piggott Osteotomy is described in 50 cases with good and satisfactory results. No other procedure in the literature gives such results. Greater care must be taken during selection of cases, technique of surgery and plaster of paris cast application. Ideal case is one with moderate deformity and symptoms referable to the increased width of forefoot without arthritis. It is an easy producer and no soft tissue procedure is needed.

References:

1. Stamm,T.T.: Surgical treatment of Hallux Valgus. Guy's Hospital Reports, 106, 273, 1957 also Guy's Hospital Reports, 1963;112; 21-26.
2. Gibson, J., and Piggott, H.: Osteotomy of the neck of the first metatarsus in the treatment of Hallux Valgus. Journal of Bone and joint surgery., 1962;44B: 349-355.
3. Helal, B. Gupta, S.K. and Gojaseni, P.: Surgery for adolescent Hallux Valgus. Acta Orthopaedica Scandinavia, 45: 271-295, 1974.
4. Kothari, S.S.: Thesis submitted University of Liverpool for award of Mch.(Ortho) 1977
5. Mitchell, C.L., Fleming et. Al.: Osteotomy - bunionectomy for Hallux Valgus. Journal of Bone and joint Surgery, 1958;40A: 41-59.
6. Wilson, J.N.: Oblique displacement osteotomy for Hallux Valgus. Journal of Bone and Joint Surgery, 1963;45B: 552-556.

XANTHO GRANULOMATOUS PYELONEPHRITIS: A CLINICO-PATHOLOGICAL REVIEW

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Abstract: *Clinico-pathologic features in a series of nine cases of xantho-granulomatous pyelonephritis are studied. There were 6 males and 3 females, ranging in the age group of 35 to 55 years with an average age of 33 years. The disease was unilateral in all the cases. There was associated urinary tract obstruction in 6 cases due to ureteric stones and renal stones. One case was due to ureteric stricture. Radiologic findings were mostly non-specific. These findings are discussed briefly in light of the salient features of this disease as published in the literature.*

Key words: *Xantho-granulomatous pyelonephritis.*

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INTRODUCTION:

Xantho-ganulomatous pyclonephritis is a form of chronic pyelonephritis characterised by destruction of renal parenchyma and presence of granulomas, abscess, collections of lipid laden macrophages (Foam cells). Although disease remains uncommon accounting for 6 per 1000 surgically proven cases of chronic pyclonephritis. It occurs from 11 months – 89 years of age but is more common in adults in 5th –7th decade [3].

Women are affected more common than men (2:1). It is unilateral but rarely bilateral [4,5]. Most patients present with pain, recurrent UTI, fever, anorexia. 73% of patients give history of previous calculous disease obstructive uropathy [9].

Materials and Methods:

This study comprises of nine cases of xantho granulomatous pyelonephritis (XGP) conducted in the Department of Pathology at SKIMS over a period of 5 years from December, 1996 to January, 2001. A total of 9 cases were identified, the medical records of 8 cases were reviewed with regard to age, sex, mode of presentation, cause of urinary tract infection, urine culture, results, side involved and therapy rendered. In the remaining one case, slides were sent to us for consultation but detailed clincial information was not provided although some information like age, sex, clinical presentation were available.

Histopathology reports and all available histologic sections as well as gross pathographs of the nephrectomy specimen were reviewed.

Pathological Findings:

Gross:

The nephrectomy specimens were small shrunken and showed marked surface scarring. Cut sections showed renal pelvis and calyces were dilated, filled with soft, bright yellow material extending upto renal parenchyma, forming nodular masses, resembling renal cell carcinoma. Almost in all cases, entire kidney was involved.

Microscopy:

The kidneys showed the features of chronic pyelonephritis with atrophy of renal parenchyma. The pelvis and renal calyces were lined by inflammatory exudate composed of diffuse lymphocytic infiltrate, plasma cell, polymorphs intermixed with aggregates of histiocytes with pale foamy cytoplasma, associated with infilltration by multineleated gaint cells. The lining epethelium of renal pelvis was ulcerated. Renal parenchyma revealed fibrosis and granulation tissue.

Discussion:

XGPN was first recognised in 1916 by Schagenhauter as distinct clinico-pathologic entity. The term XGPN was first suggested by osterline in 1944. Several individual reports as well as small and large series of cases have since been reported. This is a form of chronic pyelonephritis characterized by destruction of renal parenchyma [1-5].

Pathogenesis: It is not fully understood. Although several mechanisms have been suggested, most of the cases of XPN are associated with obstruction of the urinary tract and infection by an organism of low virulence.

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Resuls:

Table I: **Clincial Features of XGPN Cases**

Case No	*Age/sex*	*Clinical presentation*	*Cause of UTI*	*Side*	*Organisms*	*Comments*
1	*56/M*	*Right loin pain*	*Uteric stones*	*Right*	*E. Coli*	*Doing well*
2	*44/F*	*Left loin pain*	*Uteric stones*	*Left*	*E. Coli*	*Doing well*
3	*40/F*	*Left loin pain*	*Renal stones*	*Left*	*E. Coli*	*Well one year later*
4	*45/M*	*Right loin pain*	*Staghorn calculi*	*Right*	*Klebsella*	*Doing well*
5	*35/F*	*Left Loin pain*	*Renal calculus*	*Light*	*Klebsella*	*No follow up*
6	*54/M*	*Right loin pain*	*Stones at PUJ*	*Right*	*E. Coli*	*Doing well*
7	*38/M*	*Left loin pain*	*Renal stones*	*Left*	*Klebsella*	*Doing well*
8	*32/M*	*Left loin pain*	*No obstruction*	*Left*	*No growth*	*Well after one year*
9	*30/M*	*Right loin pain*	*No obstruction*	*Right*	*Not available*	*No follow up*

Table II: Radiological Findings In XGPN Cases

Case No	***Side***	***Ivp***	***Usg***	***Ct***	***Comments***
1	Right kidney	2 stones (2x1 cm)	Encho – poor mass upper pole	Low demnity	Follow up showed non-visualisation of right kidney
2	Left kidney	Stones in kidney	Encho – poor mass upper pole	Low demnity	Follow up showed non-visualisation of right kidney
3	Left kidney	Stones in kidney	Hydronephrosis	Hydronephrosis with hydroureter	Follow up showed non-visualisation of right kidney
4	Right kidney	Stones at PUJ	Hydronephrosis	Hydronephrosis with hydroureter	No other investigation
5	Left kidney	Calcification in left kidney and left ureter	Cystic and solid mass	Hydronephrosis with hydroureter	No follow up
6	Right kidney	Calcification on KUB	Mixed mass	Hydronephrosis with hydroureter	Not doing well
7	Left kidney	Calcification	Bizzarre looking mixed echogenecity	Irregular thick walled cyst	Non functioning
8.	Left Kidney	No Calcification	No obstruction	----	No other Investigation
9	Left kidney	---	---	---	---

In a majority of cases, the infecting organism is a gram negative bacillus like E. Coli, protcus mirabilies. Other include Klebslella, staphylococcus aurcus, procidentia etc. The lesions have been produced experimentally by ligating the ureter in rats followed by intravenous injection of E. Coli. About a month later, lesions closely resembling XPN may be seen [13].

Thus it appears that obstruction of urinary tract and low grade infection of the kidney may lead to development of XGPN. Obstruction of urinary tract in a majority of cases of XGPN is due to renal calculi and in some cases, obstruction is due to secondary to neoplasm involving pelvis, ureter or urinary bladder. In the present series 6 out of 9 cases as discussed before had urinary tract obstruction due to renal/ureteric stones.

It is not clear how infection and urinary tract obstruction leads to accumulation of large numbers of foaming histiocytes characteristic of XGPN.

In 1953 Friedenberg and Spujuf HJ[12], proposed an alteration of lipid metabolism with hypercholesteremia. However, this theory is contraindicated by almost constant unilateral involvement and the infrequent findings of hyperlipidemia. Arterial insufficiency was proposed by Saeed and Fine because the lesions of XGPN are relatively avascular[11].

However, other lesions associated with relative lack of vascularity such as papillary necrosis are not associated with changes of XGPN. Parson et al believed that the promileme was one of destruction of peripelvic fat by infectious process so that the lysed fat is taken by the macrophages [9].

Summary and Conclusion:

The present series has several unusual features. Firstly, the average age in our patients is 33 as opposed to 50 in most of the series. Secondly, 6 out of 9 patients in our series were male whereas, most often previously published series show a predominance of female patients. The significance of this difference is not clear although sampling error due to small number of cases in our series may be a contributing factor. Commonest side involved was left acounting for 55.5% of cases. All the pateint presented with loin pain as chief complaint. 66% of these cases had urinary tract obstruction due to stone/stricture. Radiological findings were nonspecific in all cases. A significant aspect of pathology of XGPN is that foamy histiocytes invariably present in these lesions may be mistaken for clear cells of the renal cell carcinoma. Indeed one case in our series was received for consultation after a diagnosis of renal cell carcinoma was made at some referral hospital. This could indicate that inspite of the fact that this disease has now been established for several decades, confusion with renal cell carcinoma continues to be a problem. This is because XGPN is relatively uncommon disease, so that an average Pathologist may not encounter a case in many years and thus may not be fully familiar with this condition. It is hoped that this paper will help to elucidate and high light the clinical and pathological spectrum of this interesting disease.

References:

1. Carson CC, Weinerth JL. Xanthogranulomatous pyelonephritis in renal transplant receipient. Urology 1984;23:59-61.
2. Cowley JP, Connolly CE, Hepir H, Braein O. Renal carcinoma with staghoru calculus, perinephritic abscess and Xanthogranulomatous pyelonephritis in same kidney. Urology 1983;21:635.
3. Clapton WK, Boucat HA, Dewan Pa et al. Clinicopathological features of Xanthogranulomatous pyelonephritis in infancy. Pathology 1993;25:110.
4. Malek RS, Elder JS. Xanthogranulomatous pyelonephritis: a clinico-pathological analysis of 26 cases of the literature. Journal of Urology 1978;119-589-93.
5. Mollar JC, Kristensen IB. Xanthogranulomatous pyelonephritis: clinico-pathologic study with special reference to pathogenesis. Acta Pathol Microbiol Scand (seet. A) 1980;88:89-96.
6. Goodman M, Curry T, Russel T. Xanthogranulomatous pyelonephritis: a local disease with systemic manifestation – report of 23 cases and review of the literature. Medicine (Baltimore) 1979;58:171.
7. Goulding FJ, Moser A. Xanthogranulomatous pyelonephritis with associated renal cell carcinoma. Eur Urol 1984;10:62-6.
8. Husain A, Puiqel A, Kazit T. Bilateral Xanthogranulomatous pyelonephritis. Br J Urol 1979;51:162.
9. Parson MA, Harris SC, Longstaff AJ, Urainer RG. Xanthogranulomatous pyelonephritis: a pathological, clinical and etiological analysis of 87 cases. Diag Histopathol 1983;6:203.
10. Pisccioli F, Lucianil. Association of Xanthogranulomatous pyelonephritis with small renal cell carcinoma, case report and review of the literature. Eur Urol 1984;10:62.
11. Saeed SM, Fine G. Xanthogranulomatous pyelonephritis. Am J Clin Pathol 1963;39:616-25.
12. Freidenberg MJ, Spujuf HJ. Xanthogranulomatous pyelonephritis. Am J Roentgenol Radium Thera Nucl Med 1963;90:97-108.
13. Rosip, Seltic, Carinii M et al. Xanthogranulomatous pyelonephritis: clinical experience within 62 cases. Euro Urol 1986;12:96-100.
14. Tolia BM, Selwyn AL, Futchman B et al. Xanthogranulomatous pyelonephritis: detailed analysis of 29 cases and a belief disearrion of atypical presentation. J Urol 1989;437-42.

SURGICAL ASCARIASIS IN CHILDREN

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ABSTRACT: Of the total 480 patients studied, 420 (87.5%) had round worm infestation. 300 patients had moderate to severe round worm infestation and were studied to see the severity of infestation and the results of antihelminthic drugs. 120 patients had presented with varied surgical complication due to ascariasis. Pain abdomen, vomiting and constipation were common modes of presentation. 100 patients had palpable worm masses. Eiosinophilia of more then 6 percent and haemoglobin of less then 10 gm/dl was observed in 15 and 32 patients respectively. Worm masses in plain radiographs were detected in 40 patients ultrasonography findings included worms in gut in 20 patients, signs of obstruction in 16 patients and hepatobiliary ascariasis in 10 patients. History, clinical examination, laboratory investigation and radio-imaging helped in making the diagnosis. 100 patients were managed conservatively and 20 patients underwent surgery. Bolus obstruction near ileocaecal region was the commonest surgical finding and milking of worms from ileum to coecum was the common surgical procedure adopted. Average duration of hospital stay among surgical group was highly significant statistically (p<.001).

Key words: Ascariasis, infestation, round worm

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INTRODUCTION

Ascaris lumbricoides is an intestinal parasite cosmopolitan in distribution with an overall infestation incidence of a quarter of world population [1]. A study in India has shown that stools of more than 70.0% of children have round worm ova[2]. In the valley of Kashmir the incidence of ascariasis was observed in 85.1% [3].

The main aim of this study has been to prevent unnecessary surgery in children with the help of radio-imaging techniques. It is deemed that repetitive plain X-ray and ultrasound abdominal scans help us to pick up surgical complications caused by ascariasis well in time, and enable us to continue conservative management with more confidence.

MATERIAL AND METHODS

The study was conducted in the departments of Surgery, Radio-diagnosis and Parasitology over a period of two years. 480 patients above the age of 5 months up to 14 years were studied, 300 patients (which formed non-surgical group) had moderate to severe round worm infestation, this group was studied to examine the severity of infestation and to see the effects of antihelminthic drugs used. 120 patients (which formed surgical group) had presented with varied surgical problems. Stool examination before deworming and 15 days after deworming for three consecutive days was done in all.

In addition patients with surgical ascariasis were evaluated with detailed history, thorough general and systemic examination. Haemoglobin total leucocytic count, differential leucocytic count, serum bilirubin, serum amylase and plain x-ray abdomen standing and lying down were done in all cases[4]. Contrast study of the gastrointestinal tract was done in patients who were responding to conservative treatment but had no features of visceral perforation.

Ultrasonography was performed by using 3.5 MHZ convex linear and 5.0 MHZ sector transducer on a B-mode real time scanner (Aloka, SSD, 280) sonography for biliary ascariasis was done in various scan planes including oblique subcostal, intercostal, transverse and saggital planes[5,6]. Serial sonographic examination were done to assess the exit of worms from biliary tree[5,7,8]. For intestinal ascariasis the transducer was placed longitudinally and transversely on the anterior abdominal wall with the patient lying supine and also in semiupright position[9,10].

Conservative treatment was given to all the patients with surgical ascariasis, to provide rest to the gut, relieve symptoms and signs and prevent aggravation of the disease. It consisted of intravenous fluids, nasogastric suction, antispasmodics, antibiotics and enema in selected patients[11,13]. Enema was not used in patients with suspicion

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TABLE-1: SYMPTOMATOLOGY IN 120 PATIENTS OF SURGICAL ASCARIASIS

Symptoms	No. of cases	Percentage
Pain abdomen	115	95.8
Vomiting	95	79.1
Constipation	55	45.8
Fever	25	20.8
Loose motions	18	15.0
Distension abdomen	15	12.5
Icterus	03	02.5

TABLE-2: GENERAL PHYSICAL & ABDOMINAL EXAMINATION FINDINGS IN SURGICAL ASCARIASIS

Signs	No. of cases	Percentage
Palpable worm masses	100	83.3
Tender abdomen	59	49.1
Distension abdomen	38	31.6
Dehydration	35	29.1
Pallor	32	26.6
Hyperactive bowel sounds	30	25.0
Rigidity/Gaurding	19	15.8
Drowsiness	05	04.1
Emaciation	04	03.3
Absent bowel sounds	04	03.3
Icterus	03	02.5
Tender P.R exam.	02	01.6

TABLE-3: LABORATORY FINDINGS IN SURGICAL ASCARIASIS

Laboratory Investigation	Value of investigation	No. of cases	Percentage
Eosinophill	< 6 percent	105	87.2
	> 6 percent	15	12.5
Bilirubin	< 1.5 mgm/dl	117	97.5
	> 1.5 mgm/dl	003	02.5
Amylase	< 300 IU	115	95.8
	300-1000 IU	004	03.3
	> 1000 IU	001	00.8
Hemoglobin	10-12 gm/dl	67	55.8
	8-10 gm/dl	24	20.0
	> 12 gm/dl	21	17.5
	< 8 gm/dl	08	06.6

TABLE-4: PLAIN X-RAY FINDINGS IN SURGICAL ASCARIASIS

Plain X-ray findings	No. of cases	Percentage
Bowel distension	92	76.6
Air fluid level	64	53.3
Worm masses	40	33.3

Note: More then one finding was present in a patient.

of visceral perforation, closed loop obstruction, uncertain pathology, but was used in patients who had constipation for more then two days secondary to bolus obstruction.

Surgery was performed in patients with clinical evidence of persistent intestinal obstruction, radiological evidence of increasing air fluid level, visceral perforation atomic closed loop obstruction Toxaemia out of proportion to severity of obstruction. Albendazole 200mgm below 2 years of age and 400mgm above 2 years of age stat, pyrantel pamoate 10mgm/kg body weight stat, and lavamisole as 4mgm/kg body weight stat single doses were the antihelminthic drugs used[14-19]. In conservative group drugs were given 12-24 hours after patients were free of symptoms and in operated patients 8-10 days after surgery.

RESULTS

Prevalence of ascariasis among 480 patients studied was 87.5%. History clinical examination, laboratory investigations radio-imaging response to treatment and (in operated patients) laparotomy confirmed that ascariasis was the cause of ailment in all. The results in surgical group of 120 patients are as under. 55.5% patients had acute and 39.1% had subacute intestinal obstruction, 8.3% of the patients had biliary pain. 69.1% of the patients were in the age group of 3-10 years, 63.3% were male and 36.6% female. 63% and 37% of the patients belonged to rural and urban areas respectively. Pain in and around umbilical region was present in 81.7% with colicky type of pain in 79.1% of the patients. 87% had history of passing of worms per rectum and vomiting of worms. 62.5% belonged to parents of low socioeconomic group (Table 1). The findings of general physical and abdominal examination are shown in (Table 2). Worm masses were palpable in and around umbilical region in 85% of the patients. Significant laboratory, plain x-ray and ultrasonographic findings are given in (Table 3, 4 & 5) respectively. Coiled up masses of worms which were hyperechoic and with out acoustic shadowing were noticed in fluid filled segments of gut. Linear hyperechoic shadow of ascariasis in fluid filled coils of intestines were noticed in cross section. Mean duration of disappearance and or disintegration of worm masses as seen by serial, sonographic examination of abdomen during conservative treatment was 3 days. The mean duration of exit of worms from biliary tree was 4.6 days. Contrast study examination done in 25 patients showed worm masses in 62.5% of patients.

Of the 120 patients with surgical ascariasis 100(83.3%) were managed conservatively and 20(16.6%) underwent surgery. Indication for surgery were:

Clinical evidence of persistent intestinal obstruction, gut gangrene, viscerel perforation.

Radiological evidence of increasing air fluid level, closed loop obstruction, visceral perforation, atomic closed loop bowel, intraperitoneal collection.

Toxaemia out of proportion to the severity of obstruction.

Only persistence or fixity of mass for more then 36

TABLE-5: SONOGRAPHIC FINDINGS IN SURGICAL ASCARIASIS

Sonographic finding	No. of cases	Percentage
Worm masses in intestines	20	16.6
Signs of obstruction	16	13.3
Worms in Hepatobiliary tree	10	08.3
Free fluid	09	07.5
Freely moving worms in gut	08	06.6
Local collection	01	00.8

Note: More then one finding was present in a patient.

TABLE-6: SURGICAL FINDINGS IN 20 PATIENTS

Surgical findings	No. of cases	Percentage
Bolus obstruction	16	80.0
Appendicitis	05	25.0
Perforation of viscerae	03	15.0
Volvulus	02	10.0
Gangrene of gut	02	10.0
Intussusception	01	05.0

Note: More than one finding was present in 9 patients.

TABLE-7: SURGICAL PROCEDURES DONE

Surgical Procedure	No. of cases	Percentage
Enterotomy	04	25.0
Resection and anastomosis	02	10.0
Milking of worms	09	45.0
Appendicectomy	05	25.0
Reduction of Intussusception	01	05.0

More than one procedure was done in a patient. Post operative complications were burst abdomen in 05.0%, and wound infection in 10.0% of patients.

hours was not considered on indication for surgery. Plain radiology and ultrasonography helped in subjecting 6 patients to surgery. In 14 patients both clinical and radiological evidence was the deciding factor. Surgical finding and surgical procedures performed are given in (Table 6 & 7) respectively. Effects of antihelminthic drugs are recorded in (Table 8). Mean duration of hospital stay in days of the patients managed for surgical ascariasis is given in (Table-9).

DISCUSSION

The Kashmir valley is situated at an altitude of 6,000 feet and is drained mainly by the river Jhelum, and its tributaries. Even to this day there is dearth of clean potable, piped water supply to many houses in the rural and urban areas. People use the river or lakes for washing cleaning, ablution and even drinking. The untreated refuse and ablution of many localities even in cities (including those of the many house boats) drains directly in to the river or lakes. The lack of civic amenities in most areas force people to defecate in open fields. The vegetables grown in these fields are contaminated with human faeces and ingestion of the same predisposes one to various helminthic infestation.

Ascariasis as a cause of ailment in paediatric age group has also been reported by Dasmohapatra GS et al (1971), Rehman H et al (1978) and Waller EC et al (1970)[20-22]. Most of the patients from the rural areas are in accordance with Wani (1970)[23], butare at variance with the observation of Ochoa B (1991)[24]. Pain abdomen in 95.8% patients is in accordance with the observation of Surendran N et al (1988) and Kulshrestha R (1976)[2,5]. Vomiting as the second frequent symptom in abdominal ascariasis has also been reported by Dayalan N et al (1976)[26]. Higher figures of constipation, loose motion, and distension abdomen are in consistence with Piggott J et al (1970)[27]. Icterus in 2.5% of the patients closely resembles with observations of Cremin BJ et al (1976), Mahanta J (1962) and Misra DP (1961)[28-30]. General physical and abdominal examination findings are in accordance with the findings of Rehman H et al (1978), Piggott J et al (1970), Fernando MA et al (1983) and Surendran N et al (1988)[21,27,31,2]. Eiosinophilia of more then 6 percent has also been reported by Mercer RD et al (1950) and Perlingiero JG (1957)[32,33].

Plain x-ray findings of the present study are at variance with those of Bean WJ (1965)[34], but the finding of contrast study closely resemble with Franke W (1956) and Tracey

TABLE 8: RESULTS OF ANTIHELMINTHIC DRUGS USED IN 420 PATIENTS

Drug used	No. of patients	No.of patients	Percentage cure rate
Albendazole	120	111	92.5
Pyrantel Pamoate	120	96	80.0
Levamisole	120	80	66.6

Note: All the patients with persistence of ova after treatment by pyrantel pamoate and levamisole were treated with albendazole but the results were not compared.

TABLE-9: MEAN DURATION OF HOSPITAL STAY IN DAYS OF THE PATIENTS MANAGED FOR SURGICAL ASCARIASIS IN 120 PATIENTS.

Group of patients	No.of cases	Hospital stay in days Range Mean±SD	t value	p value
Conservatively managed	100	3-36-7.55 ±7.33		
Surgically managed	20	8.43-17.98 ±7.98	5.83	<.001**
Over all	120	3-43-9.29 ±8.87		

***Highly significant*

ML et al (1950)[35,36]. The technique of ultrasonography for hepatobiliary ascariasis coincides with Kamath PS et al (1986), Khuroo MS et al (1985) and Cerri GG et al (1983)[5,6,37]. Sonographic evidence of fluid filled bowel has also been reported by Fleischer AC et al (1979) and Scheible W et al (1979)[38,39].

83.3% of the patients with surgical ascariasis in present series managed conservatively is in accordance with the study of Surendran N et al (1988)[2], but is at variance with the observations of Dasmohapatra GS et al (1971) and Cole GJ (1965)[20,40]. More patients were managed conservatively in the present study because of the general awareness of the disease, and better diagnostic facilities available now. Terminal ileum as the commonest site of bolus obstruction has also been reported by Watson FC (1920), Mathur SC (1975) and Pinus J (1982)[41-43]. In 9 patients it was possible to undo the entangled worms by gentle manipulation, manual dispersion of worm and milking them into caecum and ascending colon. In 4 patients where worms had formed into a compact mass and gentle manipulation was not possible enterotomy for evacuation of worm bolus was done above the level of obstructive mass. Only 2 patients with volvulus are at variance with the studies of Pinus J (1982), Kumar M et al (1973) and Wiersma R et al (1988)[43-35]. Surgical procedure of resection of the gangrenous segment with end to end anastomosis has also been advocated by Waller CE et al (1970), Ochoa B (1991), and Ochsner A et al (1949)[22,24,46]. Surgical findings of appendicitis, appendicitis with perforation, worms in peritoneal cavity and surgical procedure are in consistence with Waller CE et al (1970), Ochsner A et al (1949), and Sinha SN et al (1974)[22,46,47]. Appendix was the site of perforation in two patients and splenic flexure of colon in one patient. Drainage of collection, closure of colonic perforation and peritoneal lavage was done. Post-operative complications are at variance with Surendran N et al (1988) and Wiersma R et al (1988)[2,45]. None of our patient died which is totally at variance with the mortality reported by Surendran N et al (1988), Waller CE et al (1970), Mathur SC et al (1975), and Pinus J (1982)[2,22,42,43], in 14.3%, 33.3%, 11.1% and 18.8% of their patients respectively.

Finally we put the following observations on record. Ascariasis is very common in Kashmir India and can present with a grim phenamenon especially in children. Early diagnosis of its surgical complications clinically and with modern radiological techniques is mandatory prior to deciding whether it can be treated conservatively or needs surgical intervention.

REFERENCES

01. Bar-Maor J.A; DeCarvalho J.L.A.F; Chappell J: Gastrograffin treatment of intestinal obstruction due to ascaris lumbricoides. Journal of Pediatric Surgery. 1084;19(2):174-176.
02. Surendran N; Paulose M.O: Intestinal complications of round worms in children. Journal of Paediatric Surgery. Oct. 1988; 23(10):931-935.
03. Ahmed M: Incidence of helminthic and protozoal infestation in rural population of Kashmir. Thesis for MD (Medicine); University of Kashmir, 1978.
04. Isaacs I: Roentgenographic demonstration of intestinal ascariasis in children without using barium. American Journal Roentegenol Radiotherapy and Nuclear Medicine. Sep. 1956;76(3):558-561.
05. Kamath P.S; Joseph D.C; Chandran R.K; Rao S.R; Prekash M.L.S; D cruz A.J: Biliary ascariasis: Ultrasonography, endoscopic retrograde cholangiopancreatography and biliary drainage. Gastroenterology. Sep. 1986;91:730-732.
06. Khuroo M.S; Zargar S.A: Biliary ascariasis: A common cause of biliary and pancreatic disease in an endemic area. Gastroenterology. Feb. 1985;88(2): 417-423.
07. Hangloo V.K; Koul I; Dev G; Dhar U; Chrungoo R.K; Khan M: Biliary Ascariasis. Indian Journal Gastroenterol. 1989;8(4):265-266.
08. Schulman A; Loxton A.J; Heydenrych J.J; Abdurahman K.E;: Sonographic Diagnosis of Biliary Ascariasis. American Journal Radiology. 1982;139:485-488.
09. Peck R.J: Ultrasonography of intestinal ascaris. Journal of clinical Ultrasound. Nov./Dec. 1990;18:741-743.
10. Williams J.L: Fluid filed loops in intestinal obstruction. American Journal Radiology. Oct. 1962;88(4)677-686.
11. De Sa A.E: Surgical ascariasis. Indian Journal of Surgery. 1966;28:182-190.
12. Louw J.H: Abdominal complications of ascaris lumbricoides infection in children. British Journal of Surgery. 1966;53:510-521.
13. Wani N.A, Chrungoo R.K: Biliary Ascariasis: Surgical aspects. World Journal of Surgery 1992;16: 976-979.
14. Broor S.L; Bassi S.S; Broor S: Albendazole - A new broad spectrum antihelminthic for treatment of intestinal helminthiasis. Indian Journal Gastroenterol. 1986;5(4)286-287.
15. Coulaud J.P; Rossignol J.F: Albendazole: a new single dose antihelminthic. Acta Tropica. 1984;41:87-90.
16. Gazder A.J; Roy J: Albendazole suspension in the treatment of intestinal helminthiasis in children. Current therapeutic Research. March. 1987;41(3):324-327.
17. Jagota S.C: Albendazole, a broad-spectrum antihelminthic in the treatment of intestinal nematode and cystode infection. 1986;8(2):226-231.
18. Misra P.K; Pande N.K; Jagota S.C: Albendazole in the treatment of intestinal helminthiasis in children. Current Medical Research and Opinion 1985;9(8):516-519.
19. Prasad R; Mathur P.P; Taneja V.K; Jagota S.C: Albendazole in the treatment of intestinal helminthiasis in children. Clinical Therapeutics. 1985;7(2):164-168.
20. Dasmohapatra G.S; Mohanty B; Patnaik G: Clinical appraisal of 200 cases of ascariasis with special reference to surgical complications. Journal Indian Medical Association. Oct. 1971;57(8):284-287.
21. Rahman H; Singh B.B; Agarwal V.K; Srivastava A.K: "Abdominal pain in children - A clinical study". Indian Paediatrics. 1978;XV(7):583-588.
22. Waller C.E; Othersen H.B: Ascariasis: Surgical complications in children. The American Journal of Surgery. July, 1970;120:50-54.
23. Wani B.A: Incidence and symptomatology of ascariasis in Kashmir. Thesis for M.D. (Medicine), University of Kashmir, 1970.

24. Ochoa B: Surgical complications of ascariasis. World Journal of Surgery. April, 1991;15(2):222-227.
25. Kulshrestha R; George V; Vasudevan K; Dutta T: Pain abdomen - A clinical study. Indian Paediatrics. Nov. 1976;XIII(11):855-858.
26. Dayalan N; Ramakrishnan M.S: The pattern of intestinal obstruction with special preference to ascariasis. Indian Pediatrics. 1976;XIII(I):47-49.
27. Piggott J; Hansbarger C.A; Neafie R.C: Human ascariasis. American Journal Clinical Pathology. Feb. 1970;Vol. 53; Page 223-234.
28. Cremin B.J; Fisher R.M: Biliary ascariasis in children. American Journal of Roentgenology. Feb. 1976;126(.2):352-257.
29. Mahata J: Round worm (Ascaris Lumbricoides) in the common bile duct. Indian Journal of Surgery. 1962;24:605-610.
30. Misra D.P: Biliary ascariasis. Journal Indian Medical Association. Jan 1961;36(2):49-41.
31. Fernando M.A; Balasuriya; Somaratne: Effects of ascaris lumbricoides infestation on growth of children. Indian Paediatrics. Oct. 1983;20:721-731.
32. Mercer R.D; Lund H.Z; Bloomfield R.A; Caldwell F.E: Larval ascariasis as a cause of chronic eosinophilia with visceral manifestations. American Journal disease of children. 1950;80:46-58.
33. Perlingiero J.G; Gyorgy P: Chronic eosinophilia report of a case with necrosis of liver, pulmonary infilterations, anemia and ascaris infestation. American Journal Diseases of children. 1957;73:34-43.
34. Bean W.J: Recognition of ascariasis by routine chest or abdomen roentgenograms. Radiology. 1965;Vol. 94(2):379-384.
35. Francke W: Roentogenologic findings of small intestines in 100 repatriated prisoners of war. American Journal Roentgenol, Radiotherapy and Nuclear Medicine. 1956;76(1):149-152.
36. Tracey M.L; Helden GG; Bruns H.J: Roentgenographic demonstration of ascaric lumbricoides in the intestinal tract. Gastroenterology. March, 1950;14(3):428-430.
37. Cerri G.G; Jatobaleite G; Simoes J; et al. Ultrasonographic evaluation of ascaris in the biliary tract. Radiology. 1983; 146; 753-754.
38. Fleischer A.C; Dowling A.DF; Weinstein M.L; James A.E: Sonographic patterns of distended fluid-filled bowel. Radiology. Dec. 1979;133:681-685.
39. Scheible W; Goldberger L.E: Diagnosis of small bowel obstruction: The contribution of diagnostic ultrasound. American Journal of Radiology. Oct. 1979;133:685-688.
40. Cole G.J: Surgical manifestation of ascaris lumbricoides in the intestines. British Journal Surgery. 1965;52(6):444-447.
41. Watson F.C: Intestinal obstruction due to the ascaris lumbricoides. Annals Surgery. 1920;71; 757-759.
42. Mathur S.C; Gupta R.K; Gangwal K.C: Intestinal perforation by ascaris. Journal Indian Medical Association. July 1975;55(2):49-51.
43. Pinus J: Surgical complications of ascariasis. Progress in Paediatric Surgery. 1982;15:79-86.
44. Kumar M; Varier P.S; Ramachandran P: Intestinal obstruction and gangrenous necrosis due to ascariasis. Indian Journal of Surgery. 1973;35:54-55.
45. Wiersma R; Hadley G.P: Small bowel volvulus complicating intetinal ascariasis in children. British Journal of Surgery. Jan. 1988;75:86-87.
46. Ochsner A; DeBakey E.G; Dixon J.L: Complications of ascariasis requiring surgical treatment. American Journal of Diseases of Children. 1949;77: 389-407.
47. Sinha S.N; Sinha B.N: Appendicular perforation due to ascaris Lumbricoides. Journal Indian Medical Association. 1974;63(12): 396-397.

CARDIOVASCULAR RESPONSES TO TRACHEAL EXTUBATION IN NORMOTENSIVE PATIENTS; A COMPARISON WITH LMA REMOVAL

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ABSTRACT: The present study was undertaken in normotensive patients to evaluate the cardiovascular responses to LMA removal and endotracheal tube extubation. Forty patients of either sex, aged between 35-65 years and belonging to ASA status I or II undergoing elective surgical procedures were studied. They were randomly divided into two groups of twenty patients each depending on whether endotracheal tube (ETT group) or LMA (LMA group) was used. The parameters studied were HR, BP (systolic & diastolic) and rate pressure product, measured at 1,2,3,5 and 10 minutes after endotracheal tube extubation or removal of LMA. It was observed that heart rate, blood pressure (SAP and DAP) and rate pressure product increased significantly and was prolonged in the ETT group as compared to LMA group. In conclusion, LMA removal caused minimal cardiovascular changes as compared to endotracheal tube extubation.

Key words: *LMA, ETT, extubation, hemodynamic response.*

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INTRODUCTION

Endotracheal tube extubation is performed usually with the patient in a lighter stage of anaesthesia and this produces a significant increase in heart rate and blood pressure which persists into the recovery period(1,2,3). Transitory hypertension and tachycardia are probably of no consequence in healthy individuals but these responses are a matter of concern in patients with cardiovascular diseases like hypertension and coronary artery disease(4). These sympathetic adrenergic responses may lead to complications like myocardial infarction, left ventricular failure, cerebrovascular accidents and intracranial hypertension(5).

The laryngeal mask airway (LMA) is a relatively new device that fills the gap in airway management between tracheal intubation and use of the face mask(6). Besides being useful in airway management, LMA causes minimal cardiovascular responses at insertion as compared to tracheal intubation(7). We undertook this study with the objective of assessing the cardiovascular responses to LMA removal and comparing these with circulatory changes produced at extubation of the tracheal tube.

MATERIAL AND METHODS

After approval from hospital ethical committee and informed written consent, forty adult patients of ASA I and II status of both sexes, aged between 35-65 years, undergoing elective surgical procedures were enrolled in the study. Patients who were obese, pregnant, diabetic, had h/o COPD, smokers and hypertensive patients were excluded from the study. Twenty patients were intubated and formed the "ETT group"; and in twenty patients LMA was used for securing the airway and they formed the 'LMA group'. This selection for use of ETT or LMA was random. During preoperative visits, on the evening before surgery, patients were assessed and the procedure was explained to them. All patients received 0.5 mg tab. alprazolam at bed time and were premedicated with 0.5 mg alprazolam and 150 mg rantidine orally on morning of surgery.

In the anaesthetic room, an 18 guage cannula was inserted into dorsal hand vein and monitors were attached. Datex cardiocap monitor was used for continuous monitoring of EKG, heart rate, BP (systolic and diastolic) and Pace-Tech pulse oximeter was used to monitor O_2 saturation. After pre-oxygenation, all patients were induced with 5 mg/kg of thiopentone sodium, 3 mg/kg buprenorphine hydrochloride and succinylcholine 2 mg/kg was used to facilitate the endotracheal intubation or LMA insertion in a randomized manner. Lubricant jelly 2% lidocaine was applied to both ETT and LMA before insertion. Anaesthesia was maintained with 66% N_2O in O_2 and 0.5% halothane. Vecuronium bromide 0.08 mg/kg was used for muscle relaxation with top up's of 25% of loading dose used when necessary. Ventilation was provided manually using Bains co-axial circuit to maintain normocarbia. At the end of the surgery, residual neuromuscular block was

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Table 2. Showing changes in heart rate, blood pressure (systolic & diastolic) and rate pressure product in the two groups (mean±SD)

	ETT group				LMA group			
Time	Heart rate (BPM)	Sytstolic BP (mmHg)	Diastolic BP (mmHg)	Rate pressure Product	Heart rate (BPM)	Systolic BP (mmHg)	Diastolic BP (mmHg)	Rate pressure Product
Baseline	83.60 ±9.77	124.30 ±4.61	80.30 ±4.99	10403.00 ±1384.14	84.50 ±9.65	126.10 ±4.33	80.60 ±5.21	10727.80 ±1419.47
Pre-extub/ LMA removal	96.95 ±16.39	130.20 ±10.06	90.20 ±8.94	12360.30 ±2468.07	93.85 ±11.96	130.35 ±10.16	88.10 ±7.66	12280.00 ±2090.81
1 min after extub/ LMA removal	106.10 ±14.31	141.95 ±9.37	94.10 ±7.37	15189.20 ±2867.63	91.80 ±11.28*	133.50 ±7.02*	87.90 ±8.23*	12282.90 ±1895.20*
2 min after extub/ LMA removal	102.90 ±14.03	137.30 ±9.83	89.30 ±6.96	14161.30 ±2611.85	90.70 ±9.76*	131.30 ±7.40*	86.25 ±7.24	11945.45 ±1727.36*
3 min after extub/ /LMA removal	95.50 ±13.08	133.40 ±8.82	86.50 ±6.55	12807.50 ±2391.75	87.60 ±10.03*	127.30 ±7.43*	83.40 ±5.82	11165.70* ±1568.70
5 min after extub/ LMA removal	88.40 ±11.24	124.50 ±8.56	81.95 ±6.06	11054.26 ±1977.81	83.85 ±9.76	125.15 ±7.33	81.20 ±5.52	10564.65 ±1552.82
10 min after extub/ LMA removal	88.15 ±9.49	119.90 ±12.06	80.15 ±6.51	10283.00 ±1677.26	81.15 ±9.25	123.75 ±7.07	80.25 ±5.04	10152.30 ±1534.59

Comparisons are between two groups * $p < 0.05$ (sig.)

reversed with appropriate dose of neostigmine with glycopyrrolate followed by extubation or removal of LMA when the patients were awake.

Pre-induction values of heart rate and blood pressure recorded, and served as baseline values. Subsequent measurements were recorded immediately before extubation or removal of LMA and at 1,2,3,5, and 10 minutes after extubation or LMA removal. Rate pressure product was calculated (RPP = HR x SAP). The data was collected and analysed using unpaired students 't' test for intergroup comparison and paired 't' test for intragroup comparison. Values were expressed as mean±SD. p value of <0.05 was considered significant.

RESULTS

The demographic data of the patients in the two groups was comparable as shown in table 1. Changes in heart rate, blood pressure and rate pressure product are shown in table 2. It was observed that the baseline heart rate, systolic arterial pressure, diastolic arterial pressure and rate pressure products did not differ statistically in the two groups. An increase in heart rate was observed in both ETT group and LMA group at 1, 2, and 3 minutes after extubation or LMA removal, but the increase was highly significant ($p<0.001$) in ETT group and remained elevated for 3 minutes as compared to 2 minutes in LMA group.

The intergroup comparison of heart rates revealed a significant ($p<0.05$) difference at 1,2, and 3 minutes post-extubation or LMA removal. Similarly systolic blood pressure showed significant increase at 1,2 and 3 minutes after extubation ($p<0.001$) as compared to LMA group where the increase in systolic blood pressure remained for 2 minutes and was of less magnitude. Rate pressure product also showed a significant increase upto 3 minutes after extubation in ETT group as compared to LMA group.

Table 1: Showing the demographic data of the patients in the two groups.

	ETT group (n=20)	LMA group (n=20)
Age (years)	44.25±8.83	41.25±6.84
Weight (kgs)	55.80±5.79	55.30±4.81
Sex (F:M)	15 : 5	18 :2

Values expressed as mean±SD

DISCUSSION

The laryngeal mask airway was invented by Dr. Archie Brain in 1981 at Royal London Hospital, London. It has been shown to be an effective means for securing a clean airway in fasting patients for elective surgeries(7). Its insertion requires neither the visualisation of cords nor the penetration of larynx, making placement less stimulating than tracheal tube insertion or extubation(8). Therefore, during LMA insertion or removal, there is less likelihood of pressor response or coughing than with conventional endotracheal tube use.

Over the last decade, the LMA has gained widespread acceptance in the UK and USA where usage rates for general anaesthesia are in range of 30-60% of all anaesthesias administered. In contrast the device is currently used in less than 1% of patients undergoing general anaesthesia in India(9). The reasons for using an LMA are many because it has many advantages like, it is easy to insert, does not require laryngoscopy or muscle relaxants, sore throat is less and can be used in situations of the difficult intubation or failed intubation. It has been shown to cause minimal cardiovascular stimulation at insertion(10,11,12),. Very little literature is available wherein cardiovascular responses following LMA removal have been evaluated and compared with circulatory responses at endotracheal extubation.

A study conducted by Yohitaka Fujii et al found that HR, MAP and RPP increase significantly in association with extubation in healthy normotensive adults and this increase in HR, MAP, RPP was greater than after removal of LMA. These results were in agreement with our results(13).Fujii et al(14) and Sethi and Kaul (15) in their studies also reported less hemodynamic changes following LMA removal in children as compared to tracheal tube extubation.

Bidwai et al (1,2) demonstrated an increase in haemodynamic variables following extubation in normotensive patients. We also confirmed that heart rate and arterial pressure increased in association with extubation in normotensive patients. The precise mechanism responsible for these hemodynamic changes is unclear but may be associated with release of catecholamines. We also observed that increase in heart rate, blood pressure and rate pressure product was greater after tracheal extubation than those after LMA removal. This suggests that LMA has advantage over endotracheal tube in controlling the haemodynamic changes by minimizing the catecholamine release. Although rate pressure product increased significantly in both the groups in our study, but the levels were below 20,000; levels that are known to be commonly associated with angina and myocardial infarction.

We conclude that LMA should be used to substitute a tracheal tube for airway management during anaesthesia, in situations where aspiration is not a danger, to obtain a much smoother extubation and minimal cardiovascular disturbances.

REFERENCES

1. Bidwai AV, Stanley TH and Bidwai VA. Blood pressure and pulse rate response to extubation with and without prior topical anaesthesia. Canad Anaesth Soc J 1978;25:416-418.
2. Bidwai AV, Bidwai VA, Roger CR and Stanley TH. The blood pressure and pulse rate responses to endotracheal extubation with and without prior injection of lidocaine. Anaesthesiology 1979;51:171-173.
3. Edde RR. Cardiovascular responses to extubation. Anaesthesiology 1979;51:S195.
4. Miller KA, Harkin CP and Bailey PL. Postoperative tracheal extubation. Anaesth Analg 1995;80:149-79.
5. Hartley M and Vaughan RS. Problems associated with tracheal extubation. BJA 1993;71:561-568.
6. Pennant JH, White PF. The laryngeal mask airway. Its uses in anaesthesiology (Review article). Anaesthesiology 1993;79:144-163.
7. Brain AIJ: The laryngeal mask: A new concept in airway management. BJA 1983;55:801-804.
8. Pinosky M, Charleston SC. Laryngeal mask airway: uses in Anaesthesiology. SMJ June 1996.
9. Brimacombe JR, Shrapnel S and Verghese C. The laryngeal mask airway. Review Article. Ind J Anaesth 1998;42:11.
10. Braude N, Clements EAF, Hodges VM and Andrews BP. The pressor response and laryngeal mask insertion. Anaesthesia 1989;44:551-554.
11. Hickey S, Cameron AE and Asbury AJ. Cardiovascular responses to insertion of Brains laryngeal mask. Anaesthesia 1990;45:629-633.
12. Wilson IG, Fell D, Robinson SL and Smith G. Cardiovascular response to insertion of the laryngeal mask. Anaesthesia 1992;47:300-302.
13. Fujii Y, Tayooka H, Tanaka H. Cardiovascular responses to tracheal extubation or laryngeal mask airway removal in normotensive and hypertensive patients. Canadian Journal of Anaesth 1997;44:1082-86.
14. Fujii Y, Saitoh Y, Tanaka H and Tayooka H. Cardiovascular responses to tracheal extubation or LMA removal in children. Canadian Journal of Anaesth 1998;45:178-81.
15. Sethi N, Kaul HL. Hemodynamic responses to tracheal extubation in paediatric patients. J Anaesth Clin Pharma 1997;13(4):301-302.

"INHIBITION OF DIAZEPAM METABOLISM BY FLUOXETINE IN VITRO BY USING RAT LIVER MICROSOMES".

Raihana. P. Khan; S.S. Agrawal.

Abstract: The study is proposed to account for the clinically observed inhibiton of Cytochrome P450 i.e CYP2 C19 and CYP3A responsible for the metabolism of diazepam that results from the co-administration of fluoxetine. The inhibition of cytochrome P450 activity in vitro was examined in liver microsomes from male wistar rats pre-treated with fluoxetine (300 µg/kg body weight) peritoneally daily for a period of 10,20 and 30 days. The enzyme activity was found to decrease from 86% to 22% after treatment from day 10 to day 30. The total P450 content also decreased when compared with control groups after a treatment sequence of 10,20 and 30 days with inhibitor and the inhibition was found to be 24.19%, 33.73% and 43.12% respectively. The decrease in the total P450 content and enzyme activity speculates the clinically observed drug-drug interaction between these two drugs when administered concurrently in various depressive disorders.

KEY WORDS: Cytochrome P450, microsomes, Metabolism, fluoxetine, diazepam, depression

JK-Practitioner 2003; 10(1): 25-28

INTRODUCTION:

The Serotonin selective receptor inhibitors (SSRIs) are antidepressants widely prescribed for the treatment of depressive disorders, among which fluoxetine is more common. Benzodiapzepines are coadministered to treat insomnia and anxiety associated with depression in which diazepam is more common as per clinical findings.

The cytochrome P450 (CYPs) are a superfamily of heme-containing enzymes that catalyse the oxidation of a wide variety of compounds. An important role of these enzymes is to aid in the elimination of lipophillic xenobiotics, including environmental toxins and drugs. It is well known that many drugs can modulate the activity of the CYPs and therby alter the pharmacokinetic profile of co-administered drugs. (Bradley.S.Mayhem et al.

Mayhem et al)[1] Considerable effort has been expended in an attempt to predict the magnitude of *In vivo* metabolic drug-drug interaction using *Invitro* data (Rodrigues and Wong, 1997) [2]. The P450 within the P450 1,2 and 3 gene families metabolise drugs and carcinogens. The human CYP3A subfamily has CYP3A4, Which is the major enzyme in the adult liver where it is known to play an important role in the biotransformation of many drugs and endogenous steroids. Fluoxetine an anti depressant drug inhibits at least three hepatic isoenzymes P4502D6, (Lemberger *et al* 1988: Von Moltke *et al* 1994) [3,4], and probably one or more of the 2C series. The effect on 3A3/4 is suggested by the fact that fluoxetine inhibit the clearance of Alprazolam, and has been found to delay in human the clearance of diazepam, which appears to be dependent on the members of the P450 2C subfamily (Lemberger et at 1988) [3] The metabolism of diazepam is primarily hepatic and involves de-methylation involving CY2C19 and CYP3A4 and hydroxylation involving CYP3A4 and by glucronidation (Bertilsson et al 1989) [5]

In the present study inhibition of cytochrome P450 responsible for the metabolism of diazepam by fluoxetine may explain the changes in the kinetic activity of diazepam and N-desmethyldiazepam observed in our subjects.

MATERIALS AND METHODS:

Male Wistar rats 2-2½ month old were administered Fluoxetine HCL, 300 µgs/kg body weight daily for one month intrapertioneally. The rats were divided into control and treated groups having four rats in each group. The control were given vehicle i.e Tris buffer pH 7.5 intraperitoneally.

The animals were sacrificed on day 10,20 and 30 days of treatment each from control and treated group. Prior to decapitation animals were anaesthetized with ether. Livers removed and rinsed with buffer and homogenized. Microsomes were prepared after ultracentrifugation and stored at –75 ° C. Thawed microsomes were resuspended in suspension buffer and protein content determined by a method described by (Bradford MM, 1976) [6]. The by product formaldehyde during the metabolism of diazepam was determined by a modification of the method of Nash T 1953 [7] The reaction mixture consists of diazepam (3.6

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TABLE-1

S.NO.	Sample	P450 content in n moles/mg of protein	Average P450 ±SEM
1	Control microsomes treated with buffer	1.218 1.200 1.116 1.0834	1.129±0.0490

Table shows P450 content in rat liver microsome sample in control sample given vehicle only

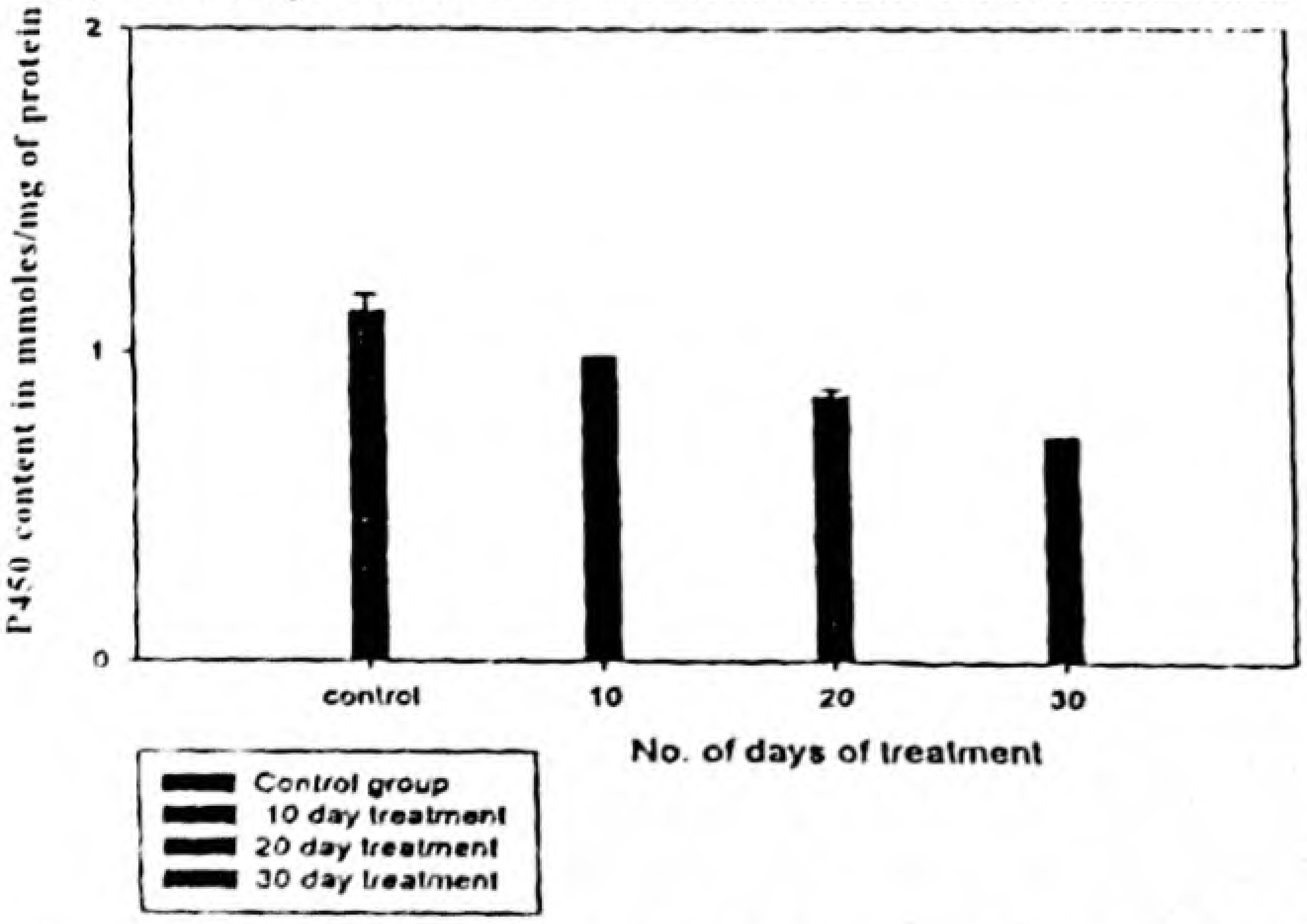
TABLE-2

S.NO.	Sample	P450 content in n moles/mg of protein	Average P450 ±SEM
1.	Rat liver icrosomes Treated,10 days n=4	0.98160 0.9932 0.9872 0.9480	0.9780±0.0101
2.	Rat liver microsome Treated, 20 days n=4	0.08299 0.8773 0.8944 0.8180	0.8550± 0.0184
3.	Rat liver microsome Treated, 30 days N=5	0.7410 0.7348 0.7355 0.7410 0.7170	0.7338± 0.004

Table shows P450 content in treated samples given fluoxetine HCl intraperitonealy for 10,20,30 days

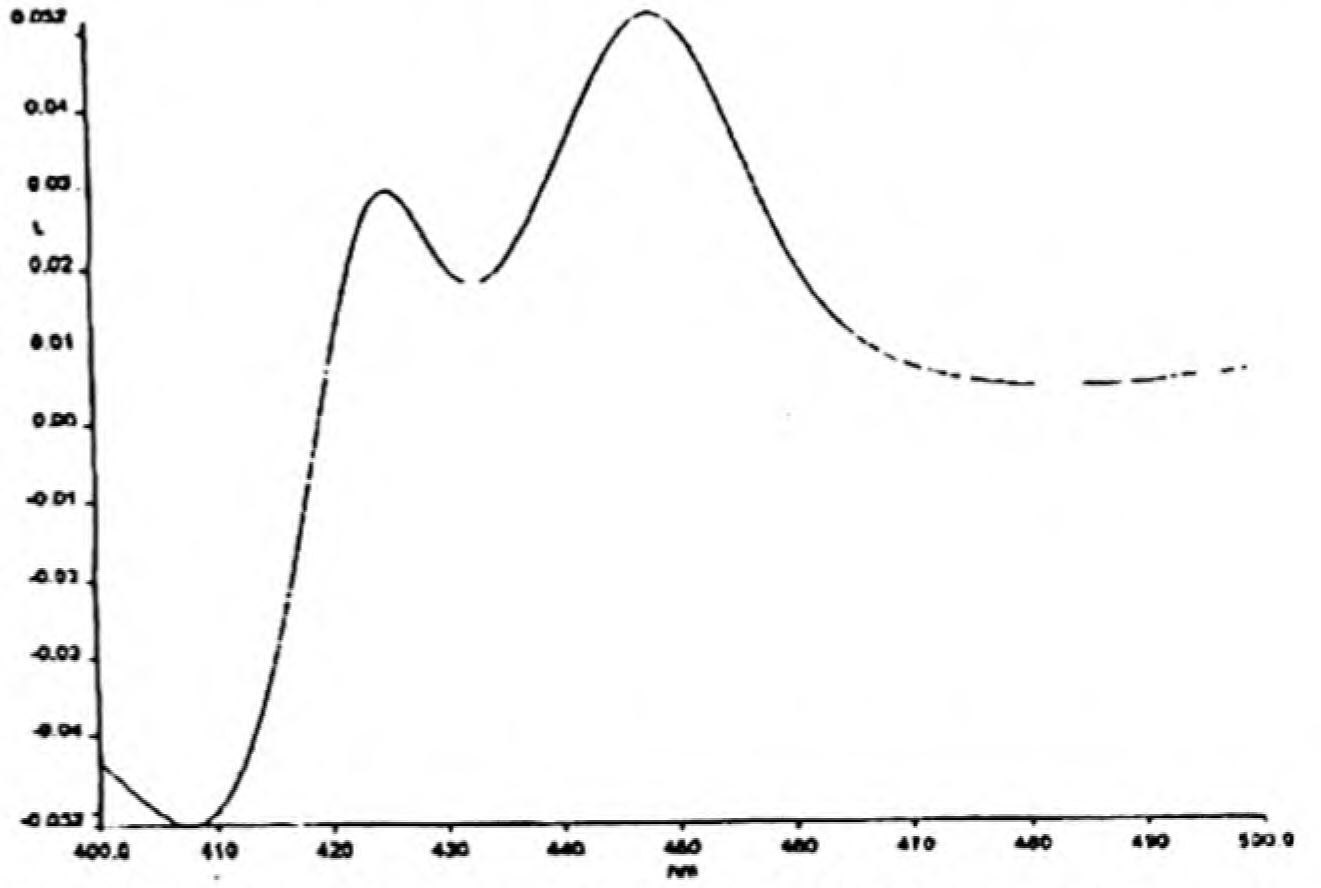
mM) as final concentration, microsomal protein conc. Of 25 and 75 μg diluted to 750 μl with assay buffer (tris-buffer pH 7.5) . The reaction mixture was taken in a test tube and was preincubated for 3 minutes, the reaction was initiated by the addition of NADPH (250 μl). The tubes were vortexed and cold TCA (500 μl) was added and the contents mixed and treated at 70° C for 20 mts, the tubes were cooled and the absorbance measured at 412 nm against reagent blank, which contains only 500μl of assay buffer, 500μl of TCA 10% and 500 μl of Nash reagent.

Fig. 1-Showing P450 content in control and treated rat liver microsomes



P450 content in rat liver microsomes treated with fluoxetine HCL fo 10, 20 and 30 days and control microsomal sample treated with vehicle only The values are mean ± SEM (n=4,4,5,4) respectively. Values are statistically significant where p=0.018 therefore $p<0.05$

Fig-2: Dithionate reduced carbon monoxide binding spectra of rat liver microsome treated with fluoxetine HCL for 30 days.



The microsomal concentration was 600ug/ml and the P450 content was 0.7410 numoles of P450 /mg of protein

Blanks containing no NADPH were run simultaneously. The blank values were subtracted from test values. All the readings were taken in triplicate. Standard tubes were run simultaneously containing varying conc. Of formaldehyde standard (4-80 nmoles i.e.5μl-100μl). The specific activity of enzyme is calculated from nmoles of formaldehyde formed /min/mg of protein.

ESTIMATION OF CYTOCHROME P450: -

Estimation of total P450 was carried out by the method described by Matusubara *et at* 1976[8].

The heme iron cytochrome P450 was first complexed with CO and then reduced with dithionate where mocrosmal concentration was kept as 0.6mg. This complex gives characteristic absorbance spectra at 450 nm and molar Ext.Coefficient has been determinated to be 106 m/cm.

The total P450 content was claculated as follows.

$$\frac{[\text{A }450\text{-A}490]\text{ test-}[\text{A}450\text{-A}490]\text{ baseline}}{0.106} = \text{nmoles of P450/ml}$$

The P450 content in the rat liver samples treated with fluoxetine for 10,20 and 30 day and control samples given vehicle only was estimated.

RESULTS AND DISCUSSION:

The total P450 content inhibited as calculated from the formula in treated microsomal samples (n=4,4,5), when compared with control samples (n=4) was found to be 24.19% in a 10-day sample, 33.73% in 20 day treated sample and 43.12% in 30 day treated sample (table 1-table 2).

TABLE-3

S.No.	No of days	% formaldehyde formed	% age activity of enzyme inhibited
1.	10	85.87	14.13
2.	20	55.52	44.48
3.	30	21.68	78.32

Table showing %age activity of enzyme inhibited having microsomal concentration of 75 μg after drug treatment for 10,20 and 30 days

TABLE-4

S.No.	No of days	% formaldehyde formed	% age activity of enzyme inhibited
1.	10	92.71	7.29
2.	20	33.72	66.28
3.	30	22.47	77.53

Table showing %age activity of enzyme inhibited having microsmal concentration of 25 μg after drug treatment for 10,20,and 30 days.

When calculated from average P450 content ± SEM.

Anova indicated a significant treatment sequence interaction for diazepam 1,5 F=15.04 and p=0.018 and the results are statistically significant.(Fig 1,2,3). The use of Hanzch,s reaction by a method described by Nash T, determination of formaldehyde concentration a by product formed during the metabolism was obtained. The formation of formaldehyde decreased from day 10 to day 30 of treatment from 86% to 22% only (fig 4,5). The decrease in formaldehyde formed reflects the activity of enzyme inhibited by inhibitor – Fluoxetine. (Table 3 – 4) shows that the formation of formaldehyde decreased at microsomal protein concentration of 25μg and 75μg at an incubation period of 20 minutes in presence of NADPH at a substrate concentration of 3.6 nmoles. Enzyme activity inhibited was calculated from the %age of formaldehyde formed as 14.13%, 44.85% and 78.32% in one group and 7.29%, 66.28% and 77.53% in other group after treatment for 10,20 and 30 days respectively.

Fig-4: Nanomoles of formaldehyde formed in control rat liver microsomes treated with vehicle only and microsomes treated with fluoxetine HCL, The microsomal protein concentration taken was 75 ugs.

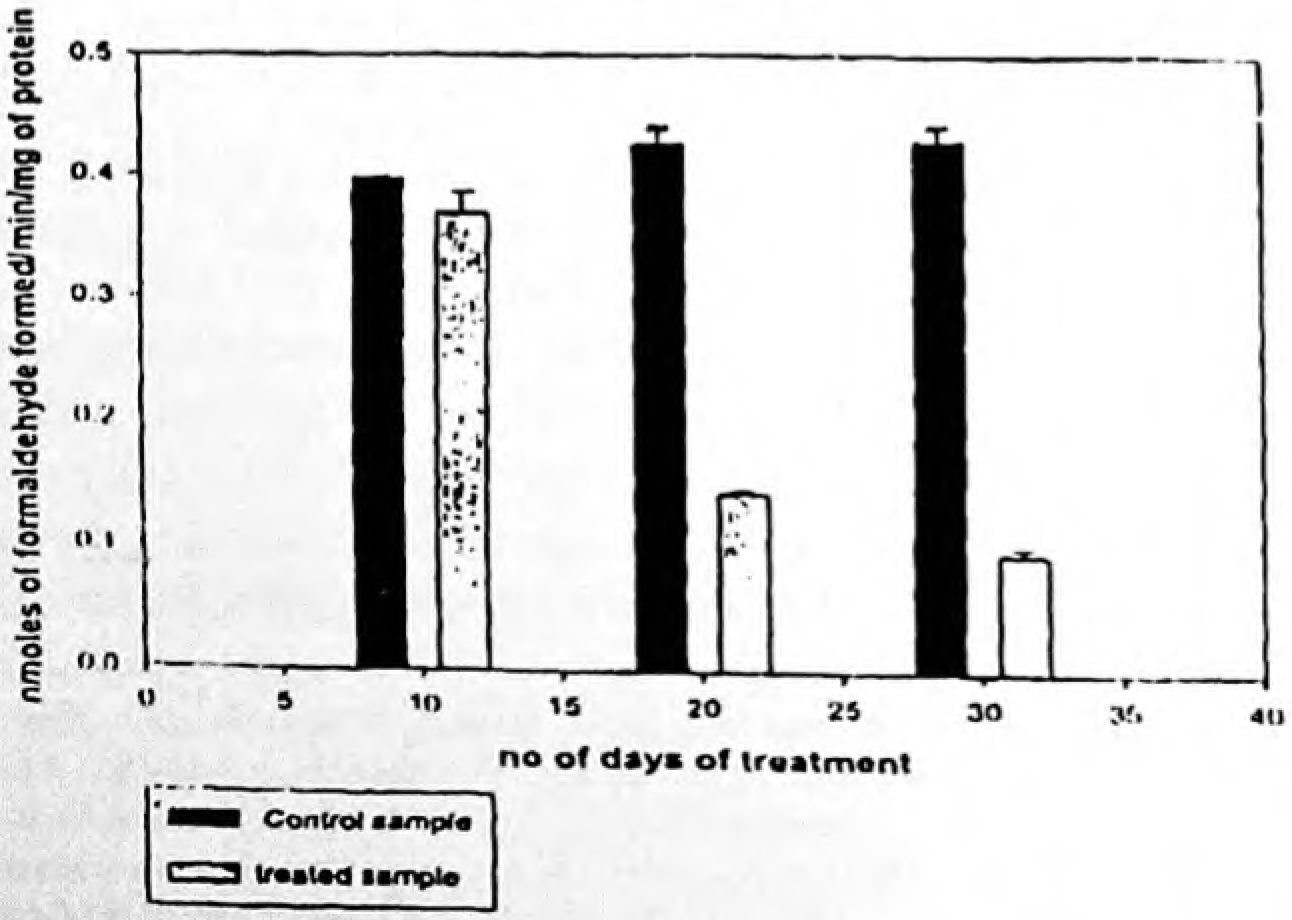
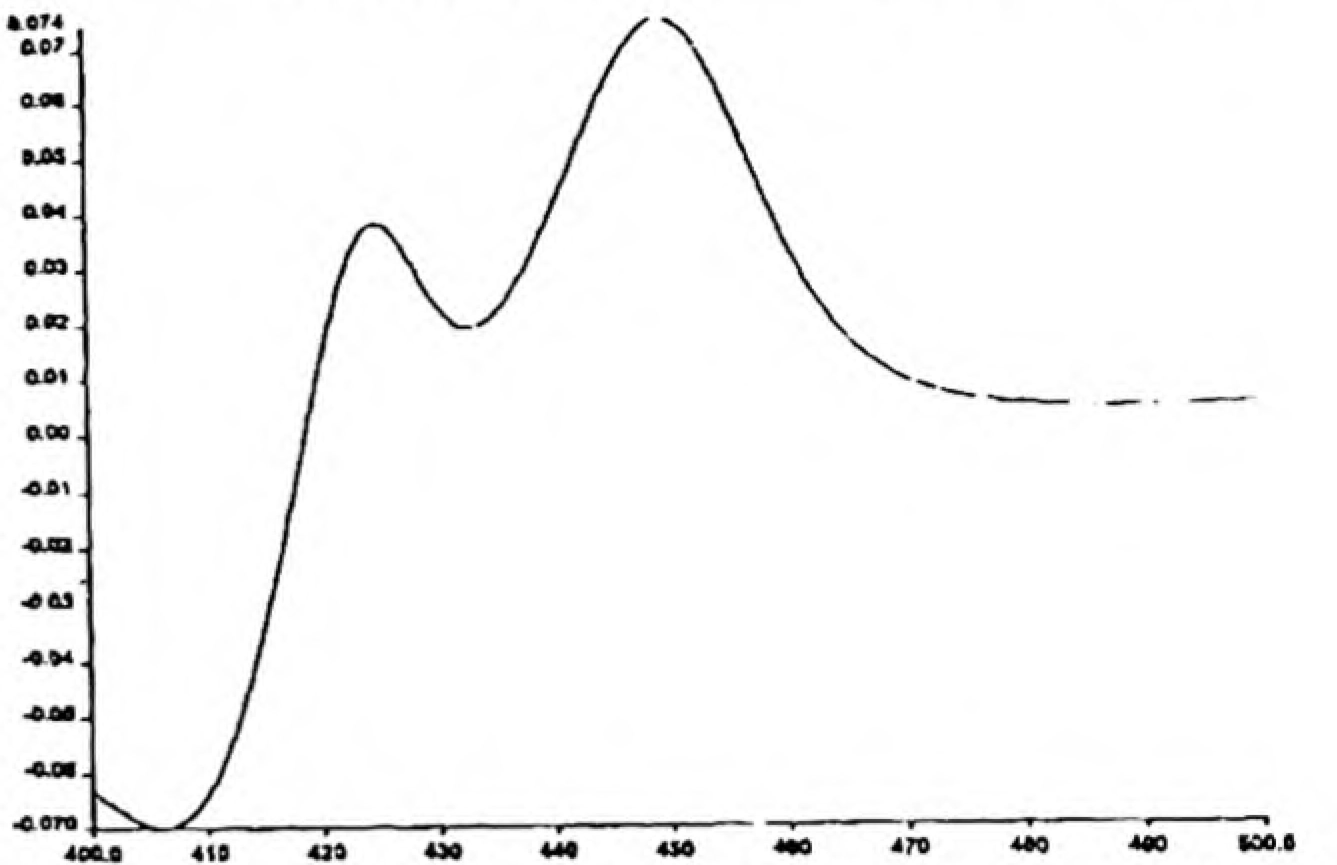


Fig-3: Dithionate reduced carbon monoxide binding spectra of rat liver microsome treated with vehicle only (Tris buffer pH7.4)



The microsomal concentration was 600ug/ml and the P450 content was 1 200 numoles of P450 /mg of protein.

This clearly depicts that diazepam demethylase reaction involved in the metabolism gets inhibited and this reflects that the reaction is dependent and medicated through cytochrome P450.

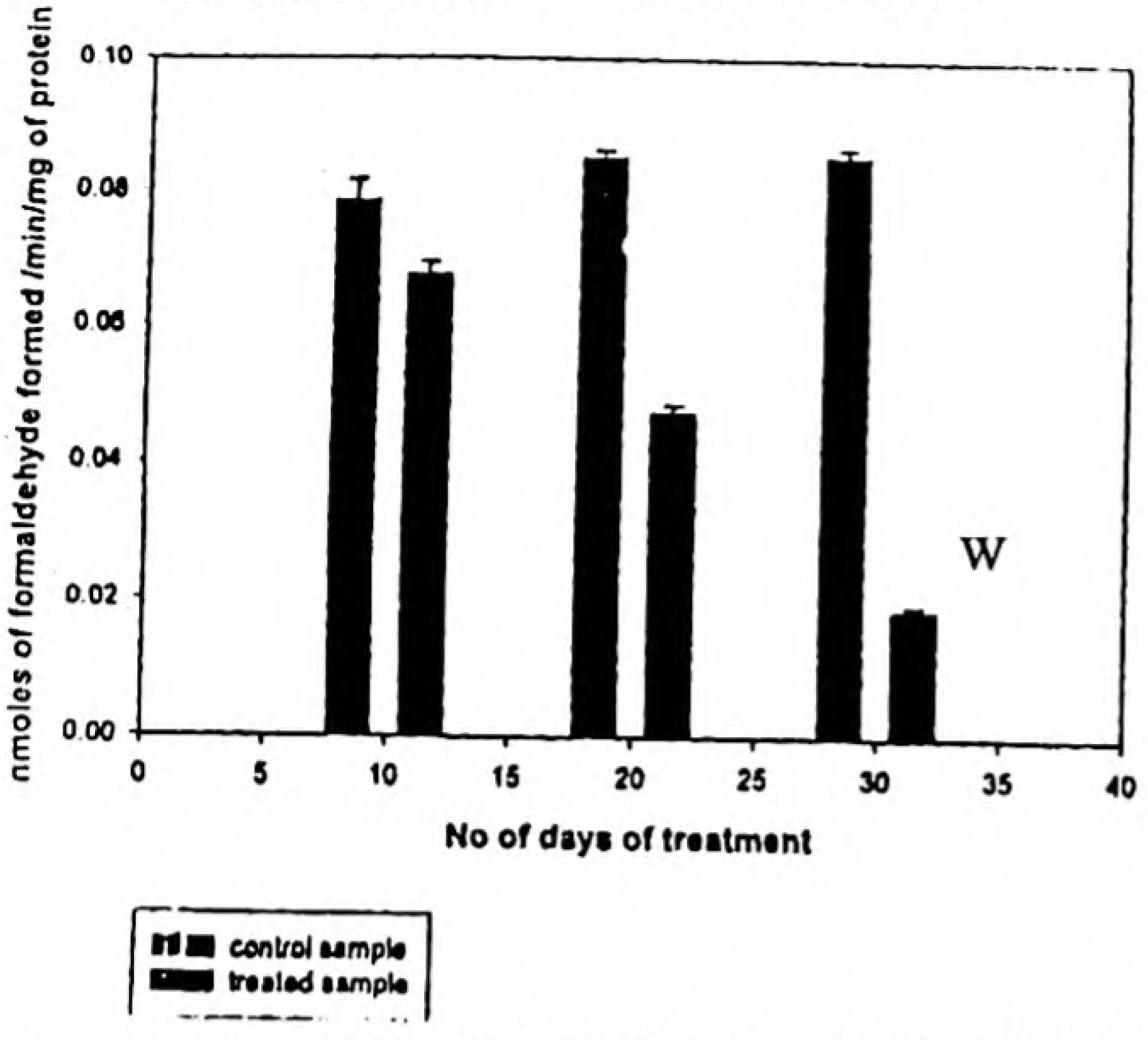
DISCUSSION

Our drugs of choice were fluoxetine and Diazepam used as antidepressant and anxiolytic respectively. The two drugs are given alone or in combination , in various depressive disorders. The drug interaction occurring during the concomitant administration has been clinically documented (Lemberger et al, 988). The increased concentration of diazepam and its metabolite in the body can lead to severe side effects like excess sedation, respiratory depression, anorexia, mood changes and muscle cramps. Diazepam is metabolized in the liver with only traces of the unchanged drug excretion in urine. Studies have shown that the conversion of diazepam to N-desmethiyhle diazepam and the 3-Hydroxylation and N-des methyhldiazepam to oxazepam is catalysed by CYP2C cytochrome identical or closely related to that responsible for S-mephenytoin hydroxylation (Bertillson et at, 1989). As fluoxetine is a potent inhibitor of CYP2C19, increasing serum concentration of benzodiazepines like alprazolam and diazepam as shown by Narnajo et al, 1999[9], it has also been found to delay in humans the clearance of diazepam that appears to be dependant on one of the members of the P4502C series (Bertillson et al, 1989)[5]

All the studies have been carried on humans and little work has been done on rats and other animals as per literature survey. The rat liver microsomes were used in this study as rats were easily available and they closely augment with the metabolism in humans. Rats provide an excellent model for the study of interaction occurring due to polypharmacy (Christopher P, Chenglis, 1992)[10]

In the present study we tried to find the extent of

Fig.5: Showing nanomoles of formaldehyde formed in control and treated rate liver microsome



Nanomoles of formaldehyde formed in control rate liver microsomes treated with vehicle only and microsomes treated with fluoxetine HCL. The microsomal protein concentration taken was 25 ugs.

inhibition of the enzyme responsible for the metabolism of diazepam by the inhibitor by measuring enzyme activity and total P450 content after treating rats with fluoxetine for a period of 10, 20 and 30 days. As per the results the total P450 content inhibited on day 30 of treatment was 43.12%, When compared with control treated with vehicle only. The enzyme activity was also measured in these slots to see the effect due to duration of treatment. This also shows that the N-demethylation reaction is mediated through Cytochrome P450. Thus we can depict that the Fluoxetine which is a potent inhibitor of CYP2D6 inhibits that CYP450 enzymes also which are responsible for the metabolism of diazepam, as per the decrease in enzyme activity from 86% to 22% from day 10 of treatment to Day 30 of treatment. The enzyme was studied at two lower microsomal concentrations and the substrate concentration was kept constant to about 3.6 mMoles, as a linear curve was not obtained at higher concentration as per protocol, might be related to decrease of stability of the enzyme substrate complex. The microsomal sample was also heated to about 70⁰ C and then centrifuged at 5000 RPM for 3 minutes to achieve thermal stability and to purify microsomal sample before proceeding as per protocol.

CONCLUSION:

The invitro studies in rat liver microsmes by the method modified in this study provided a screening method to evaluate the clinical significance of a potential interaction between fluoxetine and diazepam, due to inhibition of metabolism of diazepam by fluoxetine when prescribed for a period of one month or more. As per the clinical data collected it was concluded that physicians prefer this combination than fluoxetine-alprazolam combination which is more efficacious and less toxic in patients suffering from mixed anxiety depression and other depressive disorders. The observations stress the need of decreased use of such combinations which can other wise potentiate the side effects due to accumulation of diazepam in the body.

BIBLIOGRAPHY:

1. Bradleys S. Mahyhem, David R. Jones, and Stephen D. Hall; An invitro model for predicting in vivo inhibition of cytochrome P450 3A4 by metabolic intermediate complex formation; Drug metabolism and disposition 2000;28 (9);1031-1037.
2. Rodrigues AD and Wong SL; Application of humna livert microsmes in metabolism based drug-drug interaction; Invitro-Invivo correlation and Abbot laboratories experience; Adv. Pharmacol.1997; 43; 65-101.
3. Lemberger, L; Rowe, H; Bosnworth,JC; Tenbarge,JB : The effect of fluoxetine on the pharmacokinetic and psychomotor response of diazepam; Clinical pharmacol. Ther.; 1988 1(4);43;412-419.
4. Von Moltke, LL; Greenblatt,DJ;Corteau-Bibiboo;MM,Duan,SX; Harmatz,IS and Shader, RI:Inhibition of desperamine hydroxylation in vitrtro by SSRI antidepressant and by Qinidine and Ketaconazole; A model system to predict drug interaction in Vivo: Pharmacol. Exp. Ther.1994 : 268; 1278-1283
5. Bertillson,L; Hentroon, TK; Sanz,E; Tybring,G; Sawe,J and Villian, T; Importance of genetic factors in regulation of diazepam metabolism relationship to S-Mephentoin, but not Debrisoquin hydroxylation phenotype; Clinical Pharmacol. Ther.; 1989;45;348-352.
6. Bradford,MM;A rapid and sensitive method for the quantification of microgram Quantities of protein utilising the princi;e of protein dye binding; Anal. Biochem; 1976; 72; 248-254.
7. Nash,T; The colorimetric estimation of formaldehyde by means of Hantzch reaction; Biochem Journal; 1953;55;416-421.
8. Matushubra,T;Koike,M;Touhci,A;Tochino,Yand S, ugeno,K;Quantitiative determination of Cytochrome P450 in rat liver homogenate;Anal Biochem;1976;75;596-603
9. Naranjo,CA; Sproule,BA;Knoke,OM;Metabolic interaction of CNS medication and selective Reuptake inhibitors ;Int.clinic. Psychopharmacol ; 1999;14(2);535-547
10. Christopher,P,Chenglis;shayne,Cox.Gad;.In animal models in toxicology;1992;103-105.

ACETABULAR LABRUM TEAR WITH PARALABRAL CYST IN NON DYSPLASTIC HIP

Gulzar Ahmad Hagroo F.R.C.S.; Charles Wynn Jones F.R.C.S.; Prof. James B. Richardson M.D., F.R.C.S.

Abstract: *Although acetabular labrum tears causing disabling hip pain in dysplastic hips have been described in the literature under different headings (Paterson 1957; Alfons 1977; Grossbard 1981; Ueo 1984; Catterall 1986; Ikeda 1988; Klaue 1991; and Schnarkowski 1996). There is little mentioned about this difficult diagnostic problem in standard orthopeadic textbooks and other medical literature. This fact has also been recognised by Schnarkowski et al in their article in 1996. We are reporting a case of acetabular labrum tear (ALT) with paralabral cyst in a non dysplastic hip, which to our knowledge from a literature search has not been reported so far.*

Key words: *Acetabular Labrum Tear, Paralabral cyst.*

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Case Report

A 57 year old gentleman, an electrician by profession, who had played rugby and squash in the early part of his life sufered from progressively increasing pain in the right hip for eight years. It started as discomfort on the outer side of the right hip. He had managed to play squash for about a year or so thereafter, before it worsened and prevented him from working, walking long distance and managing home activities. The character of pain changed to a sharp catching type when he flexed and internally rotated his right hip. occassionally the pain would radiate to the leg, if pain was very severe. There was no associated back trouble. Past medical history included a fracture of the left ankle at the age of thirty one years. Clinical examination of the spine, hips and nervous system were non contributory and plain radiograph of the hips did not show any dysplasia. There, however, was a small cystic lesion in the right acetabulum superolaterally (Figure 1). The patient had no fixed flexion deformity, flexion of 100 degrees limited by pain, abduction 45 degrees, adduction and internal rotation of 5 degrees each in the right hip. Diagnostic local anaesthetic injections into right trochanteric bursa and the right hip of joint on two different occasion sduring evaluation of the problem made slight improvement in walking but, sudden catching pain during flexion and internal rotation of the afected hip persisted all along. Similar results were obtained with physiotherapy and hydrotherapy. This patient was also evaluated by the rheymatologist who gave him a clean bill of health. Isotope bone scane revealed an area of increased uptake in the upper lateral portion of right acetabulum and magnetic resonance imaging showed a cystic lesion in the same area but no labrum tear. (Figure 2) Arthroscopy of the hip revealed a tear in the labrum probably communicating with the acetabular cyst (Figure 3). Excision of the labrum tear was found to be difficult as a lot of fluid extravasated around the hip. At exploration of the right hip joint, through a standard anterolateral Watson-Jones approach, the arthroscopic finding was confirmed. There was a flap/ parrot beak tear in the posterosuperior part of labrum with a cleavage plane moving in the direction of the acetabular cyst. The latter was located by inserting a fine test drill in the cyst under image intensifier from a direct lateral approach. Gelatinous material was evacuated from the cyst and using a fine right angled probe the cyst was found to communicate with the labrum tear. An important finding was loss of hyaline cartilage just beneath the labral tear. The tear was resected and the cyst cavity packed with bone graft taken form the iliac wing. Some scoring of the anterior part of femoral head in non weight bearing area, possibly from the difficult arthroscopy, was also found. The patient had an uneventful hospital stay. He was non weightbearing on crutches for 3 months and warfarinised for the same period as he had past history of pulmonary embolism. Although this patient has no hip pain at two years following his operation and his walking is improved he still has residual discomfort on extreme rotations of the right hip. There is no catching or clicking.

Discussion

Acute catching hip pain especially in young patients

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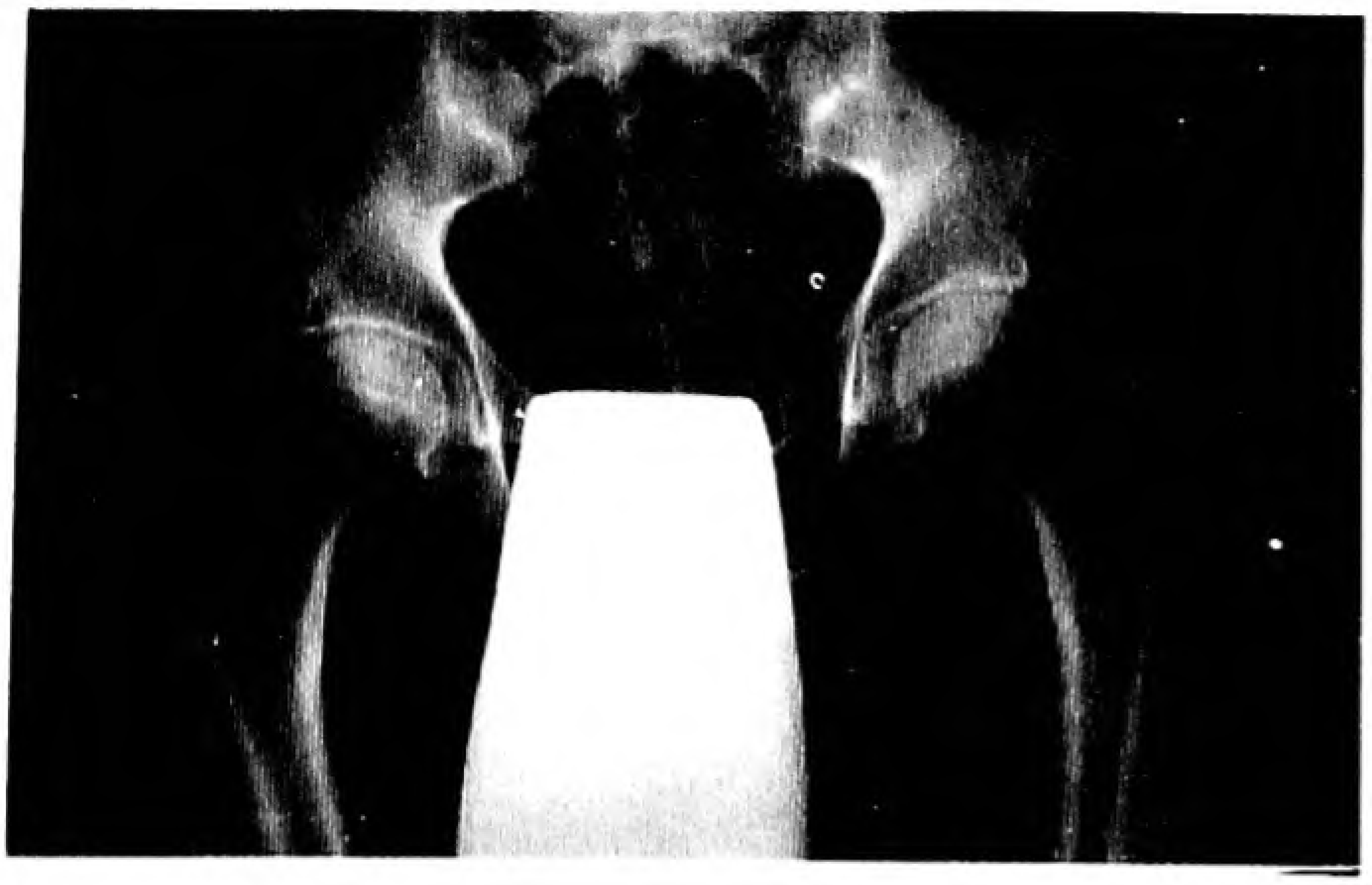

Fig. 1: Plain Radiograph of Pelvis with Hips

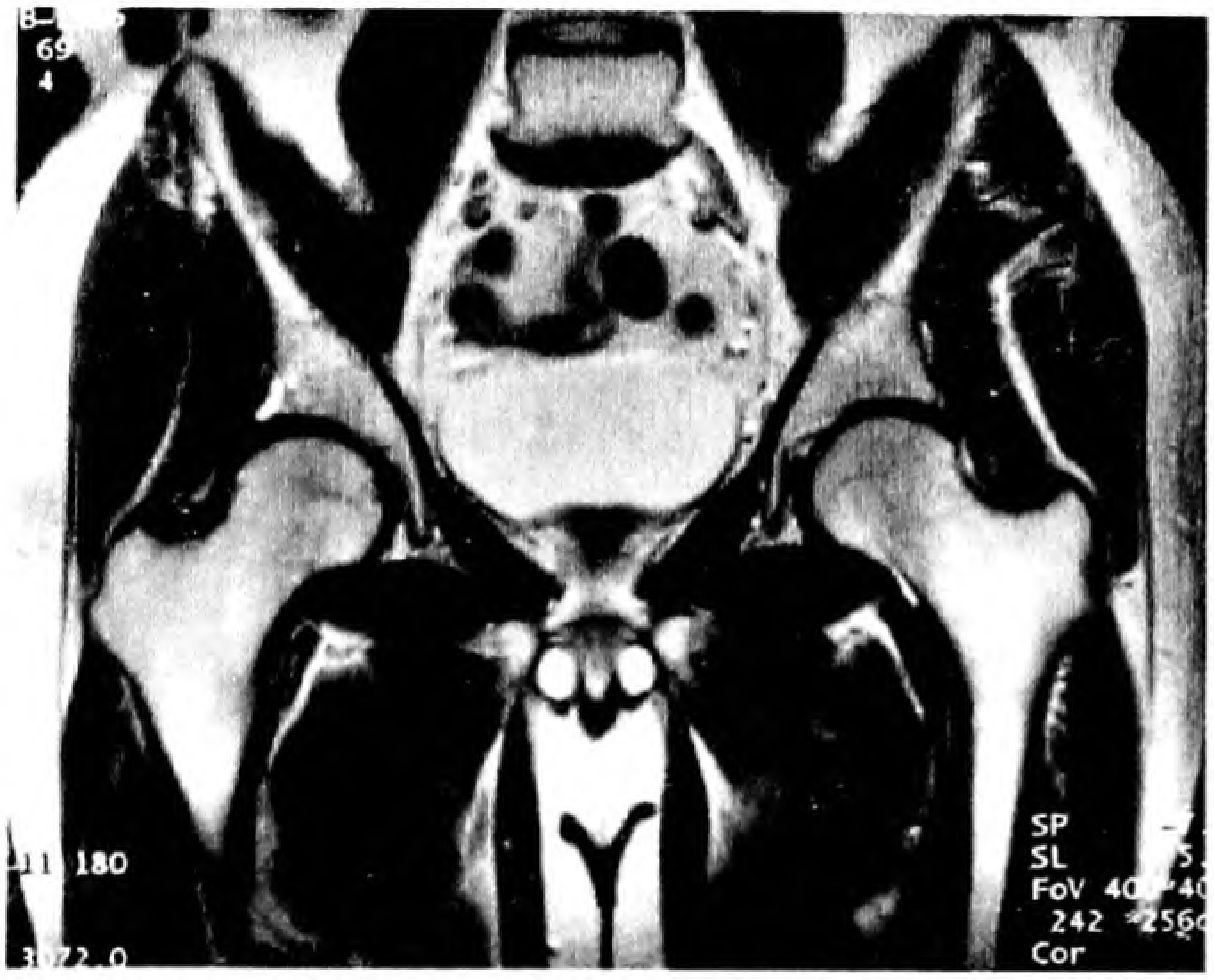

Fig. 2: Magnetic Resonance image of Pelvis with Hips

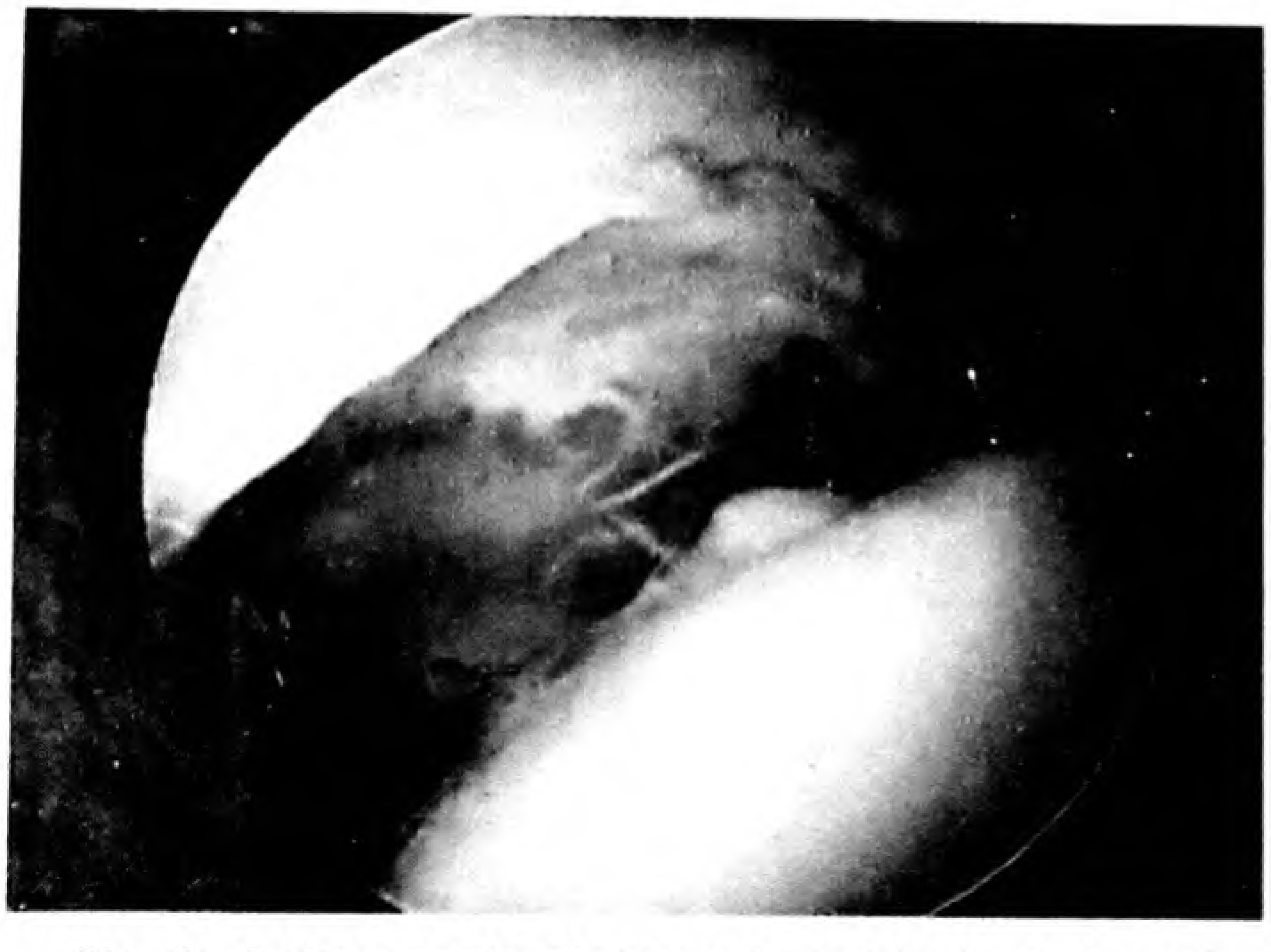

Fig. 3: Arthroscopic picture of acetabular Labrum tear

without any major history of injury to the hip or any radiological evidence of dysplasia or degenerative changes is a clinical puzzle even for experienced clinicians. It is disabling for the patient. Contrast (Gadolinium) enhanced magnetic resonance imaging (MRI has a definite role in the diangosis of ALT and / or paralabral cysts. Ikeda et al. (1988) recommends that, in doubtful cases, hip arthroscopy may be useful in both diagnosis and treatment. In our patient we found hip arthroscopy very helpful as there was no enough convincing evidence from plain radiographs and MRI scan. Ikeda (1988) and Schnarkowski (1996) are of the opinion that labrum tears and paralabral cysts are common in the weakest posterosuperior part of the acetabular labrum, which is vulnerable to repetitive minor stresses in activities of daily living and to mechanical stresses during excessive twist at sporting activities. We found ALT in our patient in the same posterosuperior region communicating through tiny hole with an adjacent small acetabular cyst. This patient was an active sportsman (Rugby and Squash player) for nearly thirty yars of his life before start of his symptoms. A tear in the acetabular labrum leads to loss of stability of the hip and joint congruity resulting in raised intraarticular pressure and joint effusion. The latter forcing synovial fluid through labrum tear into the acetabulum producing a paralabral cyst and early degenerative changes. The slowly progressing clinical history and the operative findings in our patient seem to correlate with this hypothesis. Similar observations have been made by Ikeda et al in 1988.

References:

1. Altenberg AR. Acetabular labrum tears: a cause of hip pain and degenerative arthritis. South Med J 1977;70:174-5.
2. Catterall A, Dorrell JH. The torn acetabular labrum. J Bone Joint Surg (Br) 1986;68-B:400-3.
3. Grossbard GD. Hip pain during adolescence after Perthes' disease. J Bone Joint Surg (Br) 1981;63-B:572-4.
4. Haller J, Resnick D, Greenway et al. Juxtraacetabular ganglionic (or synovial) cysts: CT and MR features. J Comput Assist Tomogra 1989;13:976-83.
5. Ikeda T, Awaya G, Shigeo S et al. Torn acetabular labrum in young patients: arthroscopic diagnosis and management. J Bone Joint Surg (Br) 1988;70-B:13-6.
6. Klaue K, Durnin CW, Ganz R. The acetabular rim syndrome: a clinical presentation of dysplasia of the hip. J Bone Joint Surg (Br) 1991;73:423-9.
7. Ueo T, Hamabuchi M. Hip pain caused by cystic deformation of the labrum acetabularare. Arthritis Rheum 1984:27:947-50.
8. Schnarkowski P, Steinbach L S, Tirman PFJ, Peterfy CG, Genant HK. Magnetic resonance imaging of labral cysts of the hip. Skeletal Radiol 1996;25:733-7.

POSTERIOR MEDIASTINAL MASS IN A 77 YEAR OLD MALE; DIFFERENTIAL DIAGNOSIS

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***Abstract:** Mediastinal masses are a common problem encountered in all age groups from infancy to older geriatric patients. Some are discovered incidentally while others may reach large size before becoming symptomatic particularly the masses located in the posterior mediastinum. The differential diagnosis of mass in the posterior mediastinum includes neurogenic tumors, cysts, esophageal lesions, infectious processes including abscesses as well as diseases of the thoracic spine. The use of CT guided transthoracic biopsy and MRI has greatly helped in the diagnosis of posterior mediastinal mass.*

***Key words:** mass, mediastinal, tumor.*

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Case report:

A 77 year old Haitian male with past history of hypertension, psoriasis and left parathyroidectomy for parathyroid adenoma came with a complaint of pain in the upper back of three weeks duration. The pain was dull, generalized over the upper back and there was no aggrevating or relieving factors. The patient had no prior history of chest disease, neoplasm or trauma. The physical examination revealed normal vital signs and there was no tenderness or limitation of motion of the upper thoracic spine. Chest and cardiac auscultation were unremarkable. There were no skin lesions, clubbing or edema. The neurologic examination was unremarkable. All laboratory work up was within normal limits except a 5-TU PPD was positive with positive skin test controls.The chest X-ray of the patient revealed widening of the mediastinum with no other specific findings.

Subsequent CT scan of the chest showed soft tissue mass in the posterior mediastinum at the level of T_4 and T_6 with vertebral body destruction. The MRI of the chest also showed paravertebral mass on both sides of T_4 and T_6 along with mild cord compression. The patient underwent CT guided aspiration biopsy which was nondiagnostic. The AFB smear was negative. The culture did not reveal mycobacterial or any fungal growth.

The **differential diagnosis** of posterior mediastinal mass includes the following:

1. Neurogenic neoplasms
   From peripheral nerves : Neurofibroma ; Neurilemoma (Schwannoma) ; Malignant tumor of nerve sheath origin (MTNSO)
   From sympathetic ganglia : Ganglioneuroma ; Ganglioneuroblastoma; Neuroblastoma; Sympathicoblastoma
   From paraganglionic cells : Pheochromocytoma ; Paraganglioma(Chemodectoma)
2. Parasitic cysts (Hydatid)
3. Meningocele (Meningomyelocele)
4. Neurenteric (Archenteric) cyst
5. Gastroenteric cyst (Pancreatic-pseudocyst)
6. Thoracic duct cyst
7. Primary esophageal lesions : Neoplasm ; Diverticula ; Megaesophagus ; Esophageal hiatus hernia
8. Hernia through the foramen of Bochdalek
9. Diseases of the thoracic spine : Neoplasms : Infectious Spondylitis (Tuberculous as well as Nontuberculous)
10. Extramedullary hematopoiesis
11. Aortic aneurysm
12. Soft tissue neoplasms (Liposarcoma)
13. Lymphoma

Discussion:

Neurogenic tumors are the most common cause of the posterior mediastinal mass. About 90% of the neurogenic tumors occur in the posterior mediastinum.[1] They represent 20% of all adult and 35% of all pediatric mediastinal tumors.[2] About 70 to 80% of neurogenic tumors are benign and half of the patients are asymptomatic. Neurogenic tumors are grouped into three categories based on their origin namely those arising from peripheral nerves, sympathetic ganglia and parasympathetic ganglia The nerve sheath tumors are most commonly seen in adults while as tumors arising from sympathetic ganglia are seen

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in children.

The tumors arising from the peripheral nerves can be either benign such as neurofibroma and schwannoma (neurilemomma) or malignant such as spindle cell sarcoma (malignant tumor of nerve sheath origin; MTNSO). The neurofibroma and neurilemoma is the most common posterior mediastinal tumor in the adult population.[3] Both are slow growing and arise from the spinal nerve root. Most patients are affected in third to fourth decade of life. Although most patients are asymptomatic, some experience pain or paresthesia from compression of adjacent structures or from intraspinous tumor extension.[4] The neurofibroma and neurilemoma is seen radiographically as sharp marginated spherical and lobulated paraspinous mass that extend from one to two posterior rib interspace and can attain large size producing pressure erosion or deformity of the ribs, vertebral bodies and neural foramina. On CT scan punctate calcification is seen and low attenuation correlates with areas of hypocellularity, cystic changes and hemorrhage within the tumor. Extension into the spinal canal produces **hourglass** or **dumbbell** configuration.[4] On MRI these tumors are having low to intermediate signal intensity on T_1-weighted images. The MRI is useful in excluding intraspinous tumor extension. The treatment of both tumors is complete surgical excision as recurrence is uncommon. About 30 to 45% of neurofibromas occur in patients with neurofibromatosis and these patients are at increased risk for malignant transformation of a neurofibroma.[5] The MTNSO arise from single or plexiform neurofibromas but rarely arise from preexisting schwannoma.[6] They are basically spindle cell sarcomas and represent the malignant counterpart of neurofibroma and neurilemoma. The male and female are equally affected in third to fifth decade of life. About half of the cases occur in patients with neurofibromatosis and most patients with neurofibromatosis and MTNSO are adolescents and young adults.[7] The common presentation of MTNSO is pain because of enlarging mass and nerve compression. The MTNSO are seen radiographically as spherical mass exceeding 5 cm in diameter. On CT scan they are seen as rounded well defined mass that locally invade the mediastinal structures and adjacent chest wall. Complete surgical resection is not possible and post-operative high dose radiation therapy is given as recurrence is common. The hematogenous spread of tumor to lungs is seen but lymph node metastasis is rare.

The tumors arising from sympathetic ganglia of posterior mediastinum include benign ganglioneuroma, malignant ganglioneuroblastoma and highly malignant neuroblastoma. The neuroblastoma can arise from adrenal gland (50%) as well as from sympathetic ganglia in the posterior mediastinum (30%).[8] The ganglineuroma is composed of mature ganglion cells and affect male and female children older than three years of age. Most of the patients are asymptomatic. Radiologically ganglioneuroma is seen as well defined oblong mass with broad base along the anterolateral aspect of spine. They can attain large size producing pressure erosion along with scoliosis and displacement of skeletal vertebra. On CT scan ganglineuroma is seen as either homogeneous or heterogeneous mass. The treatment is complete surgical excision. The ganglioneuroblastoma usually affects younger patients who are less than ten years of age. It can produce symptoms due to its large size, intraspinous extension and by local as well as wide spread metastasis of the tumor. The neuroblastoma is a highly malignant tumor seen in younger children.About 60% are younger than two years of age. It is 1.3 to 2 times common in male children. The patients can present with various neurological deficits such as horners syndrome and ataxia. The tumor can produce catecholamine and vasoactive intestinal peptides that can cause hypertension, flushing and intractable diarrhea. The patients have elevated urinary homovanillic acid and vanillylmandelic acid that help in diagnosis as well as monitoring of any recurrence of the tumor.

The tumor is seen radiographically as paraspinous mass with invasion of local structures and crossing the midline. Extensive skeletal erosion is commonly seen. On CT scan calcification of the tumor is seen and MRI shows homogenous or heterogenous signal intensity. The treatment is complete surgical resection. Chemotherapy and radiation therapy is useful in advanced stages of the disease.

The neurogenic tumors arising from the parasympathetic ganglia of the posterior mediastinum such as pheochromocytoma and paraganglioma are very rarely seen. The pheochromocytoma commonly arises from the adrenal glands in the abdomen. The **hydatic cyst** of the lung is usually seen on chest X-ray as well defined homogenous cyst (**water lilly sign**) and diagnosis is made quite easily.

The lateral thoracic meningocele is very rare entity that is seen as redundant meninges that balloons through spinal foramina. About 75% of the patients have neurofibromatosis and lateral thoracic meningocele represent the most common posterior mediastinal mass seen in these patients.[9] The CT scan and MRI can demonstrate continuity between the cerebrospinal fluid in the meningocele and that contained in the thecal sac.

The pancreatic pseudocyst can enlarge and extend along the path of least resistance to distant regions such as mediastinum, neck or scrotum.[10] The mediastinal extension is rare and posterior location is explained by the portal of entry through esoghageal or aortic hiatus or both. The CT scan and ERCP are helpful in confirming the diagnosis.

The infectious spondylitis of thoracic spine (tuberculous or non-tuberculous origin) is seen commonly in developing countries and they appear as bilateral paraspinous mass. The thoracic spine is involved most commonly in tuberculous spondylitis (Potts disease). The classic pattern of tuberculous spondylitis is destruction of intervertebral disk and in advanced stage of the disease there is a formation of paravertebral abscess. Early treatment with anti-tuberculous therapy can prevent neurological complications. The unusual causes of infectious spondylitis include disseminated histoplasmosis, coccidiodomycosis and blastomycosis in the endemic area.[14] The actinomycosis of the spine also can cause paraspinal abscess and is usually the result of contiguous abdominal or pelvic spread.[11]

The ***extramedullary hematopoietic tumors (EMHTs)*** are associated with chronic congenital hemolytic anaemia such as thalassemia, hereditary spherocytosis or sickle cell disease. These tumors can be asymptomatic presenting as unilateral or bilateral paraspinal masses that contain fat and hematopoietic tissue.[12] While EMHTs usually occur in patients with severe chronic anemia, such lesions must be considered in the differential diagnosis of posterior mediastinal mass in patients without clinically evident anemia.[12] The EMHTs may rarely complicate chronic myeloproliferative disorders.[15]

The ***soft tissue tumor of posterior mediastinum*** such as liposarcoma is a very rare entity and patient can have symptoms of cough, dyspnea, chest pain and cachexia due to the pressure effect of the tumor.[13] The treatment is surgical excision of the tumor.

The ***primary mediastinal lymphomas*** may affect any mediastinal compartment and present as nodal enlargement. The mediastinal involvement is made by cross-sectional imaging studies such as CT scan and MRI. Both Hodgkins disease and non-Hodgkins lymphoma can affect the mediastinum and manifest as a primary mediastinal lesion or as a generalized disease.

In view of our patients age the following differential diagnosis were highly considered : infectious spondylitis, lymphoma, neurogenic tumor, soft tissue tumor and myeloproliferative syndrome of EMHTs. The patient underwent right posterolateral thoracotomy along with excision of posteriormediastinal mass and spinal reconstruction surgery. A frozen section diagnosis of fibroadipose tissue with focal chronic inflammation was made and the stains were negative for AFB or any fungal growth. The subsequent pathology report came as chronic fibrosing osteomyelitis of $T_5 - T_8$. The culture for the mycobacteria organism is still pending. The patient was started on fourdrug anti-tuberculous drug therapy.The patient condition improved and back pain decreased. Our case illustrates that in an elderly geriatric patient with posterior mediastinal mass the infectious spondylitis should be considered one of the differential diagnosis in addition to any neoplastic etiology.

CONCLUSION:

Although the differential diagnosis of posterior mediastinal mass is extensive, the roentgenographic and clinical signs permit a considerable narrowing of the possibilities. The CT scan and MRI should be a part of the evaluation of such lesions. Early diagnosis is important in order to prevent permanent neurological complications. Despite extensive preoperative evaluation, some patients have to undergo thoracotomy in order to confirm the diagnosis. The physician should always keep in mind all the possible differential diagnosis when a patient comes with a posterior mediastinal mass.

References:

1. Benjamin SP, McCormack LJ, Effler DB et al : Primary tumors of the mediastinum. *Chest 1972;62:297-303.*
2. Azarow KS, Pearl RH, Zurcher R et al : Primary mediastinal masses. *J Thorac Cardiovasc Surg 1993;106:67-72.*
3. Reed JC, Kagan HK, Feigin DS : Neural tumors of the thorax : subject review from the AFIP. *Radiology 1978;126:9-17.*
4. Akwani OE, Payne WS, Onofrio BM et al : Dumbbell neurogenic tumors of the mediastinum. *Mayo Clin Proc 1978;53:353-58.*
5. Aughenbaugh GL : Thoracic manifestations of neurocutaneous diseases. *Radio Clin North Am 1984;22:741-56.*
6. Thomas JE, Piepgras DG, Scheithauer B et al : Neurogenic tumors of the sciatic nerve. *Mayo Clin Proc 1983;58:640-47.*
7. Hope DG, Mulvihill JJ : Malignancy in neurofibromatosis. *Adv Neurol 1981;29:33-56.*
8. Swanson PE : Soft tissue neoplasms of the mediastinum. *Semin Diagn Pathol 1991;8:14-34.*
9. Alrabeeah A, Gillis DA, Giacomantonio M et al : Neurenteric cysts- A spectrum *J Pediatr Surg 1988;23:752-54.*
10. Shivakumar D, Dourgoutis DG, Shah MR : Posterior mediastinal mass lesion. *Chest 1984;85:683-84.*
11. Voisin L, Mejjad O, Krzanowska C, Defivés T, Cambon MC, Le Loet X : Spinal abscess and spondylitis due to actinomycosis. *Spine 1998;23:487-90.*
12. Montpreville TV, Dulmet ME, Chapelier RA, Dartevelle GP, Verley MJ : Extramedullary hematopoietic tumors of the posterior mediastinum related to asymptomatic refractory anemia. *Chest 1993;104:1623-24.*
13. Yang R, Elliston L, Peterson R, Sahmel R : Dysphagia and cough in a patient with a posterior mediastinal mass. *Chest 1987;92:529-30.*
14. Maugire HJ : Osteomyelitis and infections of the prosthetic joints. *Harrisons Principles of Internal Medicine 1994;13:558-61.*
15. Beckman EN, Oehrle JS : Fibrous hematopoietic tumors arising in agnogenic myeloid metaplasia. *Hum Pathol 1982;13:804-10.*

DEPRESSION AS THE PRESENTING FEATURE OF TUBEROUS SCLEROSIS

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Abstract: *Tuberous sclerosis is a rare autosomal dominant disorder in which neurological involvement manifests in the form of seizures, mental retardation, affective and behavioural derangements. We report a case, whose main presenting feature was major depression, not mentioned in previous literature.*

Key words: *Seizures, Mental retardation, Tuberous sclerosis, Depression.*

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Introduction

Tuberous sclerosis (also called Bourneville's disease or Brustifield - Wyatt disease) is a congenital disorder of autosomal dominant variety in which a variety of lesions due to hyperplasia of ectodermal and mesodermal tissue occur in skin, nervous system, heart, kidney and other organs.[1,2,3] Among the neurological manifestations seizures and mental retardation are the commonest features.[2,3,4] In addition hydrocephalus, dementia, hyperkinesis, spastic weakness, choreoathetosis and behavioural derangements are reported.[1-4] Interesting in our case, is that patient presented with features of major depression, and subsequent work up proved the patient suffering from tuberous sclerosis. Depression as a feature of the disorder has not been mentioned in the previous literature, and this prompted us to report this case.

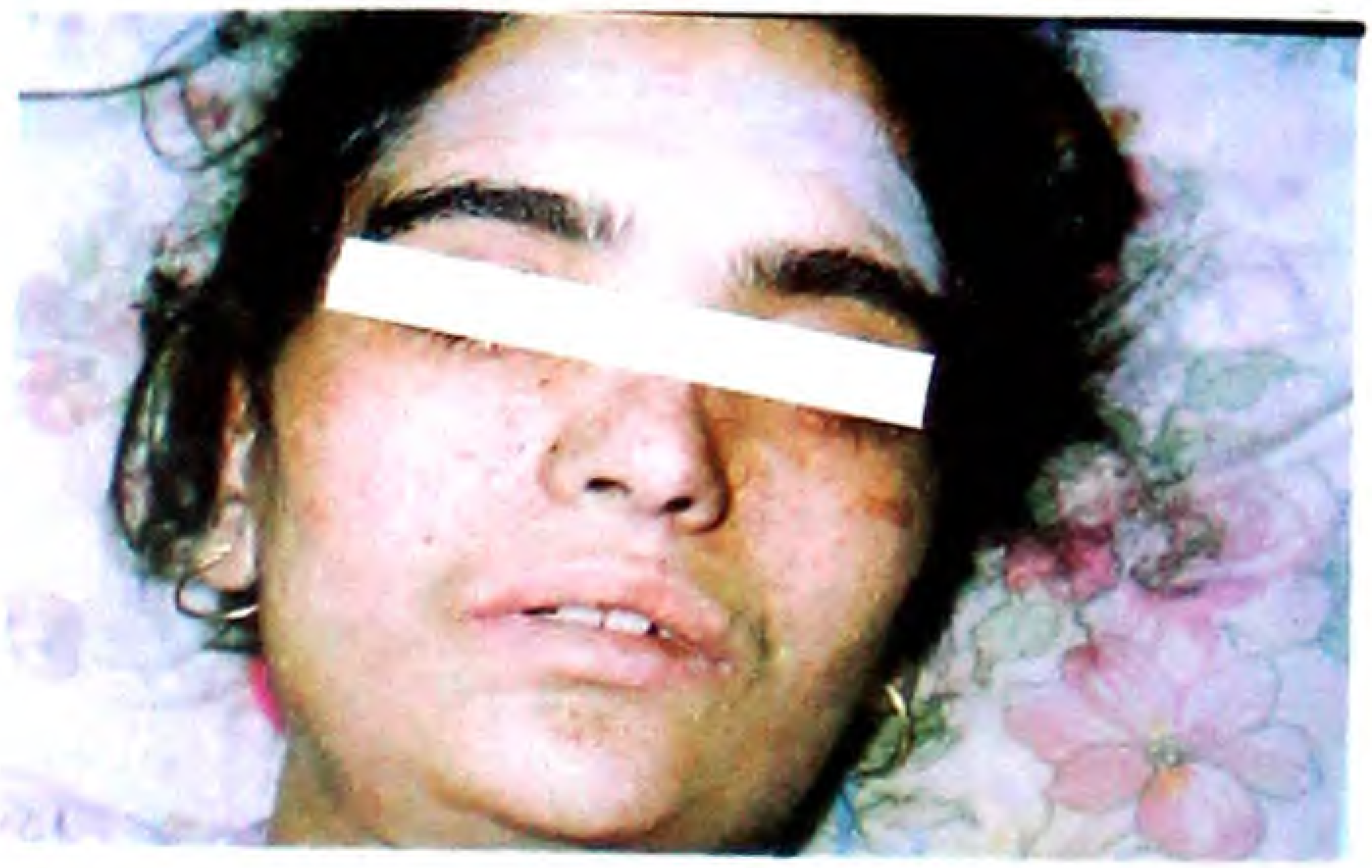

Fig.1: Adenoma Sebaceum Lesions

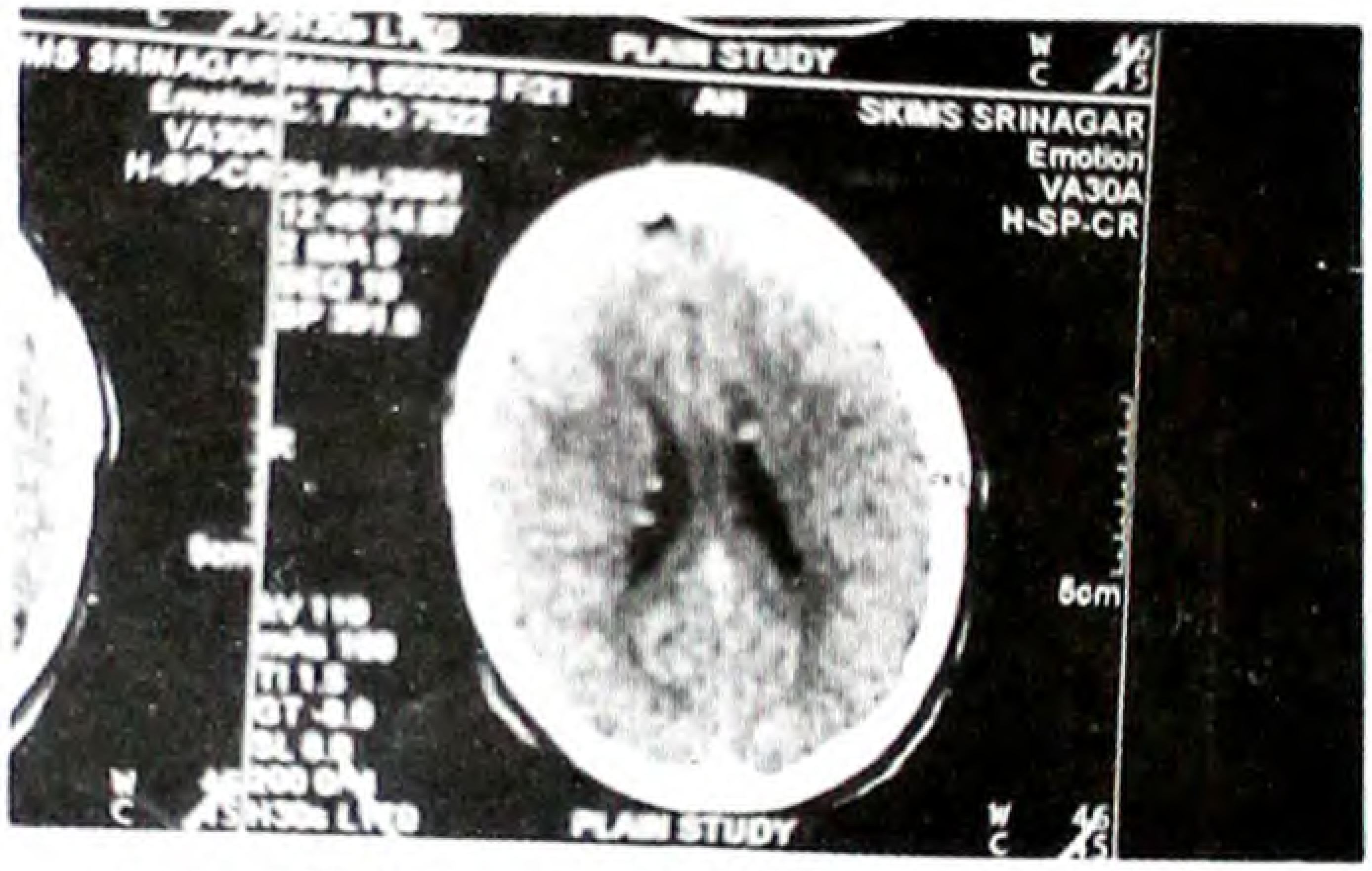

Fig.2: CT Brain showing Calcified Subependymal tubers.

Case Report:

A 28 year old female, product of a consanguineous marriage and second in birth order, got admitted to our department with history of fever and behaviour disturbance one weak after marriage. She was noticed to have decreased communication with members of the new family and other relatives. She developed lack of interest, loss of sleep and appetite, mental deterioration and episodes of agitation as narrated by her husband. Psychiatrist was consulted by us, who labelled her as having major depression, put her on antidepressants and advised to do a detailed medical evaluation to rule out the presence of any organic illness. Her past history was elicited from all possible relatives, by which we came to know that she was having few episodes of generalized tonic - clonic seizures since last 14 years for which she was put on anticonvulsants (including phenytoin), otherwise there was nothing significant in her developmental, family and the past history. On examination

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she was conscious, slow to react to verbal commands and was febrile (temperature 101 - 103 °F, intermittent), with pulse of 108 - 124 beats / min and blood pressure of 130/70 mmHg. On her face, she was having reddish - brown papular lesions on cheeks, also extending from nasolabial folds to the chin. A few scattered erythematous papular lesions were also found over forehead (Fig 1). Another skin lesion was an 'ash-leaf' shaped hypopigmented lesion in her right lumbar region. These features wer consistent with the diagnosis of tuberous sclerosis as per the opinion of dermatologist, and was evaluated on the lines of same. In her general physical and systemic examination no other abnormality was found. On investigating she was having leucocytosis (13000/mm^3) with predominance of polymorphs. Rest of all baseline investigations including CSF and blood cultures revealed no abnormality. She was put on broad spectrum antibiotics and fever subsided. Ultrasonographic study of the abdomen revealed a solid mass 3.2 x 4 cms in relation to the upper pole of right kidney. CT scan of brain revealed numerous calcified subependymal tubers (Fig 2). On the basis of skin lesions, subependymal tubers and renal mass she was diagnosed as a case of tuberous sclerosis. For renal mass she was planned for open biopsy but the patient died of sudden cardiopulmonary arrest before that.

Discussion

Among the cutaneous lesions sebaceum, ash-leaf shaped hypopigmented macules, shagreen patches and depigmented nevi are commom[1,3] common findings of tuberous sclerosis, as were also found in our case. Rhabdomyomas of the myocardium, and angiomyomas of kidney, liver, adrenals and pancreas have also been reported.[1-5] Our case had a solid mass in relation to right kidney, but unfortunately tissue diagnosis of the same couldn't be achieved as the patient died prior to that. The commonest neurological feature of tuberous sclerosis and patients with later onset can present in late childhood, adolescence or adult life with tonic clonic, complex partial, or myoclonic seizures[1,2] and these seizures are always the most reliable index of the cereberal lesions.[1] There exists a very close relationship bettween the age of onset on one hand and the severity of epilepsy and the development of dementia, hyperkinesis and autistic features on the other.[4] Mental functions continue to deteriorate slowly. Spastic weakness or mild choreoathetosis of the limbs can also occur.[1] In addition to obstructive hydrocephalus and mental retardation, other features including constant crying, muttering, rocking and swaying movements and digital mannerism have been observed.[1,4] Focal neurological deficits can occur as a result of giant - cell astrocytomas. Our patient's main presentation was depression, in addition to other manifestations of tuberous sclerosis which is not reported anywhere in literature so far and needs to be taken into consideration. Since our patient could not survive, fwe can not comment on the prognosis of disorder. It has been found that prognosis depends upon the severity of mental, psychiatric and epileptic manifestations, and 40 to 50 percent of patients have been found living normal life.

References:

1. Victor M, Ropper AH. Developmental diseases of the nervous system. In: Adam's and Victor's - Principles of Neurology. 7th Ed. McGraw Hill, New York, 2001;1050-1105.
2. Harding AE. Neurocutaneous disorders and degenerative diseases of the spinal cord and cerebellum. In Walton J (ed.). Brain's Diseases of the Nervous Syste, 10th ed, Oxford University Press, 1993;426-452.
3. Sagar SM, Irrael MA. Tumors of the Nervous System. In: Fauci AS, Braunwald E, Isselbacher KJ et al (eds.). harisons Principles of Internal medicine 14th ed McGraw Hill, New York 1998;2398-2409.
4. Neville BGR, Collins JE, Surtees RAH. Paedriatric neurology. In: Walton J (Ed) - Brain's Diseases of the Nervous System. 10th Ed, Oxford University Press, 1993;453-77.

UNILATERAL CRYPTOPHTHALMOS

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ABSTRACT: *Cryptophthalmos, a very rare congenital anomaly of the eye, is characterized by skin passing continuously from the forehead to the cheek over a malformed eye. Here we report a case of unilateral Cryptophthalmos in a 10 day old neonate in view of the rarity of this condition.*

Key words: *Cryptophthalmos, Congenital*

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INTRODUCTION:

Cryptophthalmos is a very rare, probably autosomal recessive condition in which the lid folds fail to separate in the embryo resulting in a continuous sheet of skin from the forehead to the cheeks covering the malformed eye. It has been reported infrequently in the literature with fewer than 100 cases reported and only two cases have been presented in China [1,2].

CASE REPORT

A 10 day old male neonate, product of a consanguinous marriage was brought to the eye OPD of SMHS Hospital by the parents complaining of absence of left eye. On examination, there was absence of left eyebrow, both left upper and lower lids and a sheet of skin was seen covering the area of the left eyeball **[Fig. 1]**. Movement of the eye was occasionally visualized beneath the skin. On palpation a normal feel of an eyeball with closed lids was felt. The right eye ball and appendages were normal. Systemic examination revealed no other abnormality. Family history was non-contributory.

The patient was operated upon, revealing a malformed, cystic eye ball with no differentiation of ocular structures **[Fig. 2]**.

DISCUSSION

Cryptophthalmos is a rare congenital anomaly characterized by an uninterrupted continuity of the skin of the forehead with that of the cheek resulting in total ablephorn. It was first reported in 1872 by Zehender and Manz [3]. It is thought to occur mostly on a sporadic basis, but a recessive inheritance has been suggested and occurs bilaterally twice as often as it does unilaterally and there seems to be a slight preponderance of males. Predilection for the left eye in unilateral cases has been reported [4].

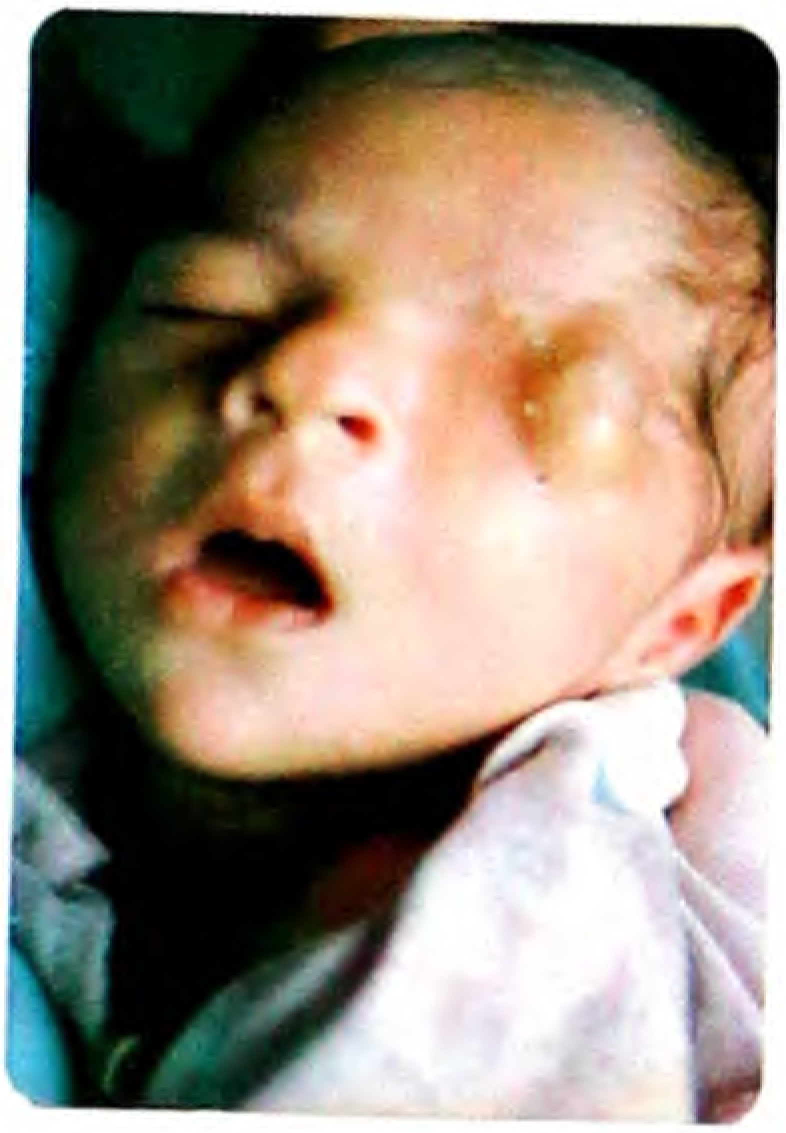

Fig.1: Clinical Photograph of a 10day old neonate with unilateral cryptophthalmos

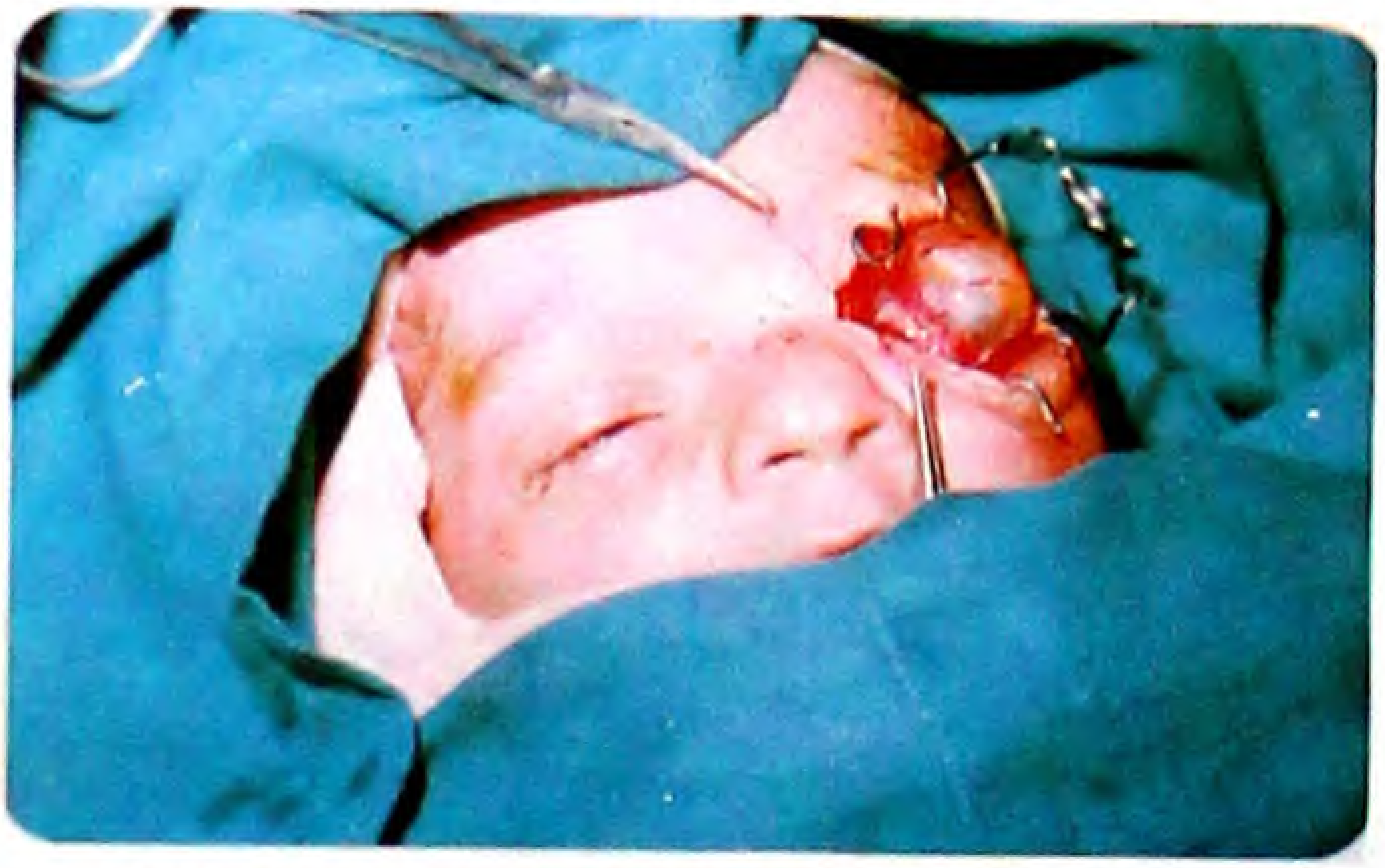

Fig.2: Operative photograph showing disorganized globe.

Cryptophthalmos is divided into three types, the complete variety, the incomplete variety and the symblepharon variety. The complete variety is the most common type. The eyelids do not form and the eyelid skin

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grows continuously from the forehead to the cheek, covering the underlying globe. The globe is usually disorganized.

The incomplete variety presents with facial skin fusing to the medical aspect of the globe with no eyelid structures in that area. The lateral eyelid is present and normal.

Finally, the congenital symblepharon variety presents with fusion of the upper lid skin to the superior portion of the globe. The eyelid in this area is absent. There may be a small amount of normal eyelid laterally.

Cryptophthalmos is usually associated with many other congenital anomalies, including mental retardation, nasal anomalies, ear anomalies, cleft lip and palate, irregular dentition, genitourinary abnormalities, cardiac malformations, meningoencephalocele, abnormal hairline, umbilical hernia, anal atresia, ankyloglossia, laryngeal atresia and syndactyly [5].

There are syndromes associated with Cryptophthalmos and these include. Fraser's syndrome, Cryptophthalmos syndactyly syndrome, malformative syndrome with Cryptophthalmos and Cryptophthalmos syndrome [6].

The patient discussed represents the complete variety of Cryptophthalmos. Because the eye is disorganized and the conjunctival sac is absent, recovery of sight and reconstruction of the lid and conjunctival sac by surgery are impossible. Because of these drawbacks a kerato prosthesis may be the only alternative.

REFERENCES

1. Chou CH. Cryptophthalmos. A case report Chin Med J (Engl) 1928; 42 : 907.
2. Fan MC. Cryptophthalmos. A case report Chin Med. J (Engl) 1959 ; 79 : 172.
3. Duke Elder WS. System of Ophthalmology. London, Kimpton. 1964 ; 3 : 827-33.
4. Gupta VC, Sen DK. Unilateral cryptophthalmos. Indian J Ophthalmol. 1990 ; 38(2) : 97-9.
5. Zhang HC. Cryptophthalmos : A report of three Sibling cases. Br. J. of Ophthalmol 1986; 70(1) : 72-4
6. Kabra M, Gulati S, Ghosh M, et al. Fraser Cryptophthalmos Syndrome. Indian J Pediatr 2000 ; 67 (10) : 775-8.

WEBER'S SYNDROME ASSOCIATED WITH GIANT ANEURYSM OF THE POSTERIOR CEREBRAL ARTERY

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Abstract: *A young female was clinically diagnosed as a case of Weber's Syndrome and was further evaluated by CT, MRI and MR - Angiography (MRA). These investigations revealed a partially thrombosed giant aneurysm of the right PCA which is a rare entity.*

Key words: *Weber's syndrome, Giant Aneurysm, Posterior Cerebral Artery (PCA), CT, MRI, MR - Angiography (MRA)*

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Introduction

An aneurysm on the posterior aspect of the circle of Willis can lead to Weber's syndrome (AKA superior or oculomotor alternating hemiplegia) by involvement of the third cranial nerve as it passes through the brainstem. Deficits include ipsilateral ptosis, pupillary dilation, lateral strabismus and absent light reflex (IIIrd nerve involvement) and contralateral paralysis of the arm and leg (cortico-spinal involvement).

Case Report

A 30 year old female with a drooping right upper eyelid and a right sided headache and left sided weakness worsening over 2 days was admitted to the Department of Medicine at S.M.H.S. Hospital, Government Medical College, Srinagar in March 2002. She had no past occular problems, and medically she had a unremarkable past history. A diagnosis of Weber's syndrome (ipsilateral third nerve paresis and contralateral hemiparesis due to involvement of the pyramidal tract) was considered and the patient was referred to the Department of Radiodiagnosis and Imaging where CT Scanning (HiSpeed-FXi, GE), MRI and MR-Angiography (Symphony, Siemens) were performed.

CT demonstrated a large round well-defined denisty in the posterior circulation with a peripherally organizing thrombus (fig.1). No evidence of rupture of the aneurysm was seen.

MRI showed a hyperintense rounded structure with artifacts, characteristic of low-velocity flow (Fig 2,3 & 4) with a staged laminated intensity representing thrombus.

MRA showed change of a flow signal in posterior cerebral artery (PCA) indicative of pathologic widening of the PCA (Fig. 5,6,7). The dimensions of the structure measured 4.9 by 2.4 cms.

A partially thrombosed giant aneurysm of the right PCA was confirmed on the basis of these findings.

Discussion

An intracranial aneurysm is a small, thin walled outpouching or dilatation of one of the large blood vessels that supply the brain. A giant intracranial aneurysm is defined as an aneurysm larger than 2.5 cm in diameter. Treatment of large (2-2.5 cm) and giant aneurysms has traditionally been associated with a higher morbidity and mortality than smaller lesions.

Aneurysms of the posterior cerebral artery (PCA) make about 1% of all intracranial aneurysms[1,6,8,9]. PCA aneurysms are more typical of its proximal segments[5,6,8]. They have some specific morphologic signs, which differentiate them from aneurysms of other locations. As per Ferrante and collegues[5] they are more frequent in young people (average 38 years), whereas the average age of patients with aneurysms of anterior areas of the arterial circle is 50-60 years. In our case, the patient was 30 years old.

One more typical feature is a high rate of large and giant aneurysms (23%-50%) among PCA aneurysms in comparison with 3-5% among aneurysms with other locations)[4,7,8,10]. Our patient had a giant aneurysm measuring 4.9 by 2.4 cms almost qualifying as a supor-giant aneurysm (>5cms)..

Seventy to seventy-five percent of patients with giant aneurysm present with mass effect usually manifest by visual failure, cranial nerve dysfunction, hemiparesis, seizure or headache. Pia H.[8] reports a high frequency (27%) of vision impairment in PCA aneurysms; oculomotor disorders and hemianopsia are most frequent. Ciceri E.[2] observed 5 patients (25%) with oculomotor and vision disturbances due to compression of the oculomotor nerve and chiasm. Our patient presented with Weber's syndrome

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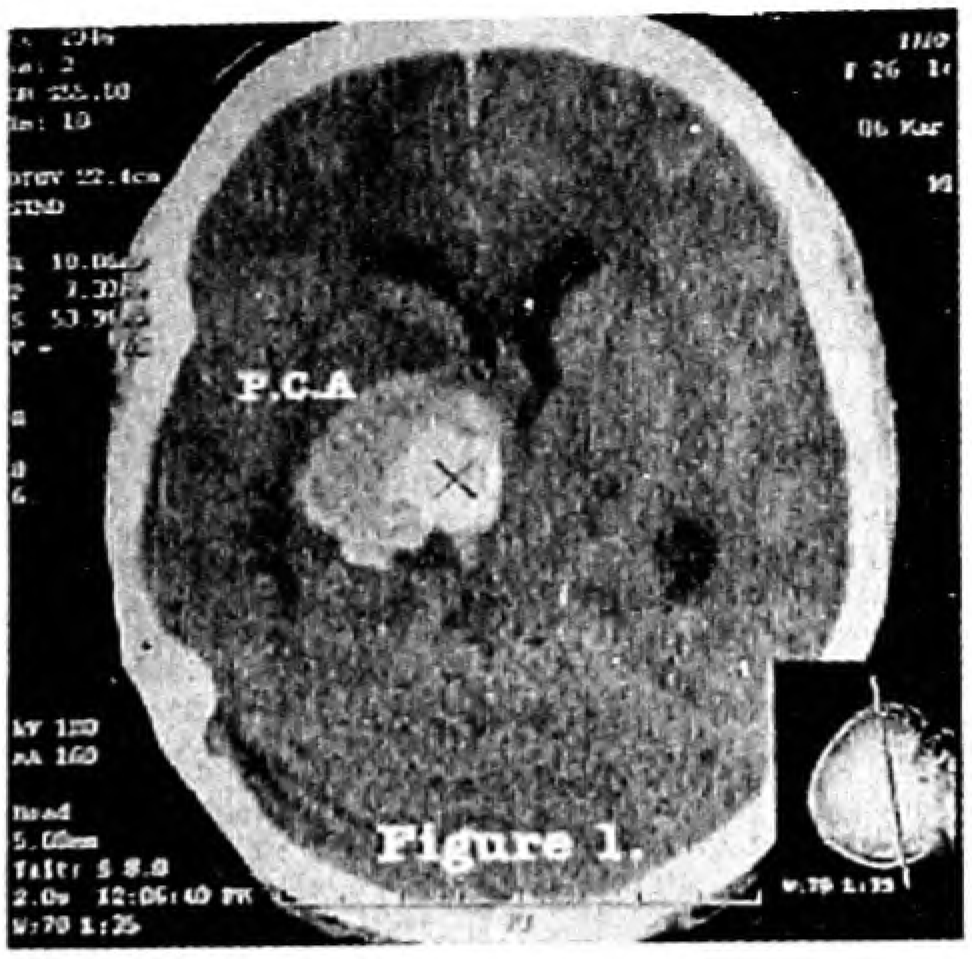

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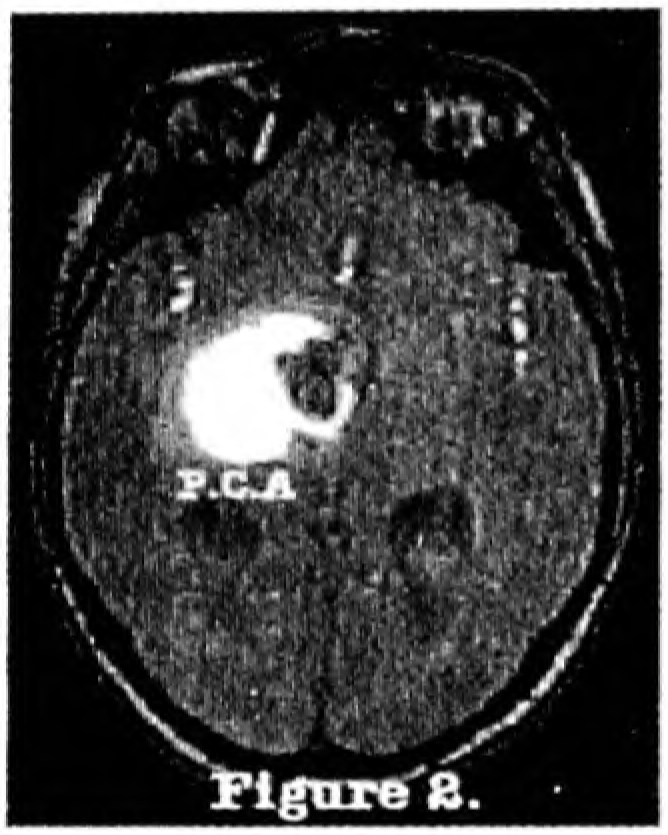

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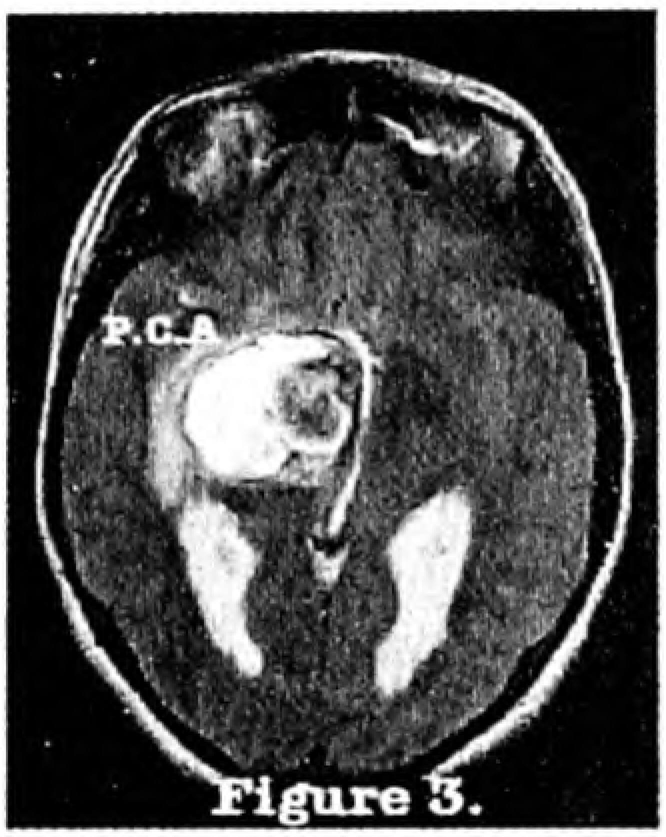

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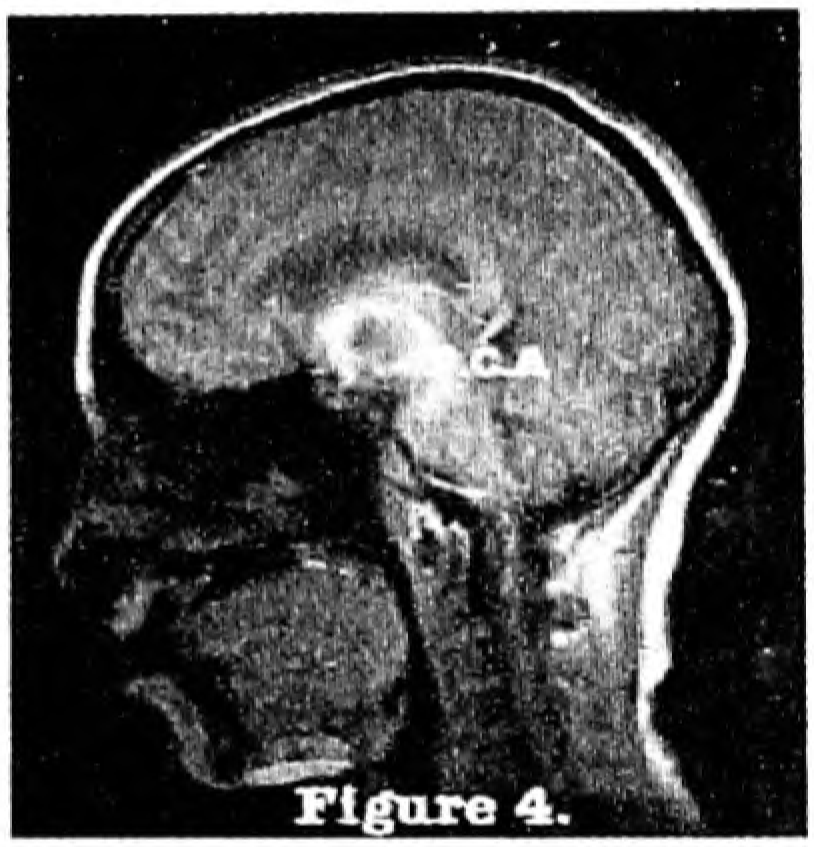

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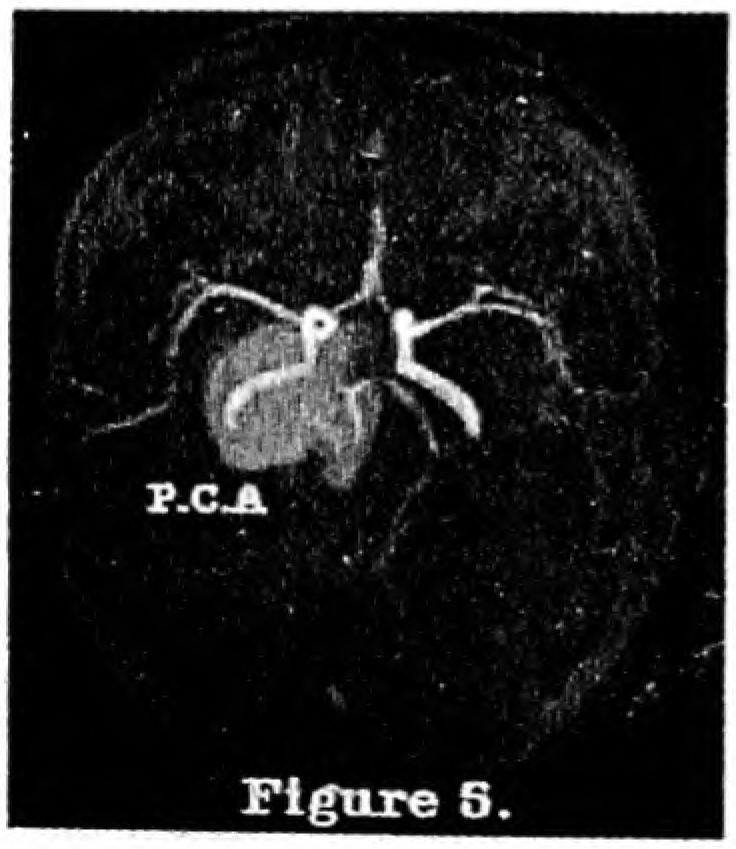

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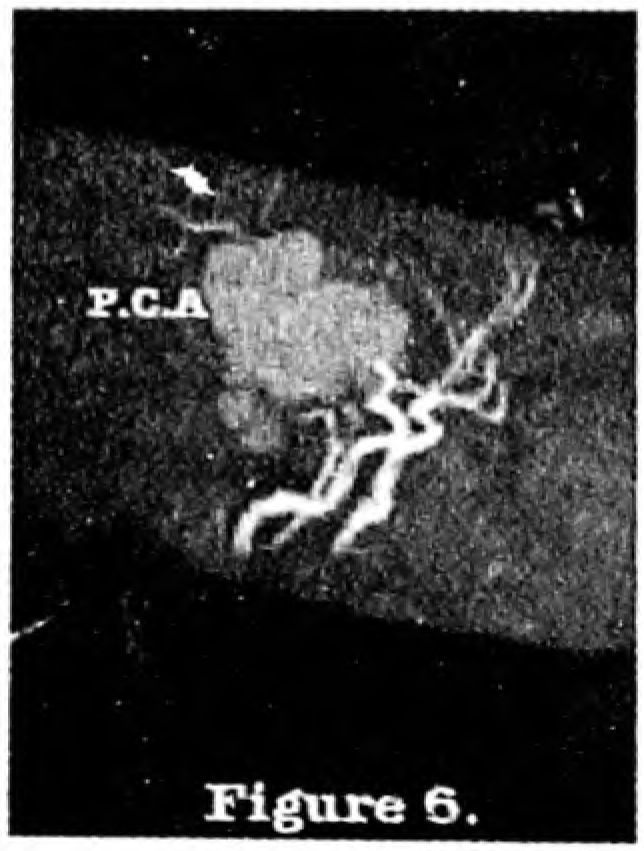

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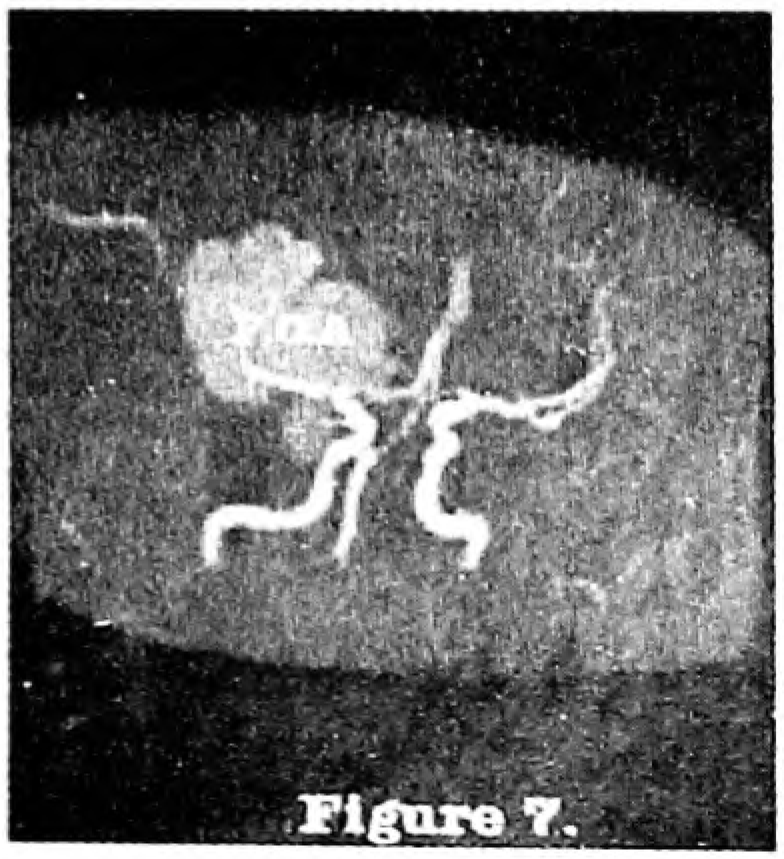

Figure 7.

(ipsilateral third nerve paresis and contralateral hemiparesis). Thrombosis and stroke due to blood clot formation within the aneurysm and subsequent distant embolus, has been reported in approximately 2-5% of patients with giant aneurysms. Thrombus formation within the aneurysm was observed in our patient.

Subarachnoid hemorrhage is one of the most frequent manifestations of PCA aneurysms and is seen in 50-80% of cases[5,6,8]. In our patient there was no evidence of rupture of the aneurysm, despite its remarkable size.

According to Brennan J.W. and Schwartz M.L.[1] while there is insufficient data to support a standard regarding surgical repair of unruptured intracranial aneurysms, surgery is appropriate for the management of giant aneurysms. Surgical techniques include Microsurgical clipping, Embolisation or Arterial occlusion and bypass.

As far as we know this is the first reported case of this nature in the Kashmir Valley.

References:

1. Brennan J.W., Schwarz M.L. Recommendations for unruptured aneurysms Neurosurgery. 2000;47(12):1359-1372.
2. Ciceri E.F., Klucznik R.P., Gorssman R.G. et al. Aneurysms of the Posterior Cerebral Artery: Classification and Endovascular Treatment AJNR. 2001;22(1):27-34.
3. Drake C.G. Giant intracranial aneurysms: experience with surgical treatment in 174 patients. Clin/Neurosurg 1979;26(1):12-95.
4. Drake C.G. Giant posterior cerebral aneurysms: 66 patients In: Drake CG, Peerless SJ, Hirnesniemi JA, eds. Surgery of Vertebrobasilar Aneurysms: London, Ontario, Experience on 1767 patients. New York: Springer Verlag. 1996;230-248.
5. Ferrante L., Acqui M., trillo G. et al. Aneurysms of the posterior cerebral artery: do they present specific characteristics? Acta Neurochir (Wien). 1996;138(10):840-852.
6. Gerber C.J., Neil-Dwyer G. A reivew of the management of 15 cases of aneurysms of the posterior cerebral artery. Br. J. Neurosurg. 1992;6(4):521-527.
7. Lempert T.E., Hallbach V.V., Higashida R.T. et al. Endovascular treatment of pseudoaneurysms with electrolitically detachable coils AJNR 1998;19(6):907-911.
8. Pia H.W., Fontana H. Aneurysms of the posterior cerebral artery. Locations and clinical pictures. Acta Neurochir (Wien). 1997;38(1):13-35.
9. Sakata S., Fujii K., Matsushima T., et al. Aneurysms of the posterior cerebral artery; report of elevent cases-surgical approaches and procedures. Neurosurgery. 1993;32(2):163-167.
10. Yasargil M.G. In: Microneurosurgery. Vol. 2. Stuttgard: George Thieme Verlag. 1984;260-269.

DIFFERENT FOREIGN BODIES IN URINARY BLADDER

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Abstract: Foreign bodies in urinary bladder are not uncommon. However very little published series are available although not being a fatal disease can lead to serious complications like chronic cystitis, stone formation or fistula formation. We present three cases of different foreign bodies with different etiology which were removed successfully.

Key Words: Foreign bodies, bladder, Heamaturia.

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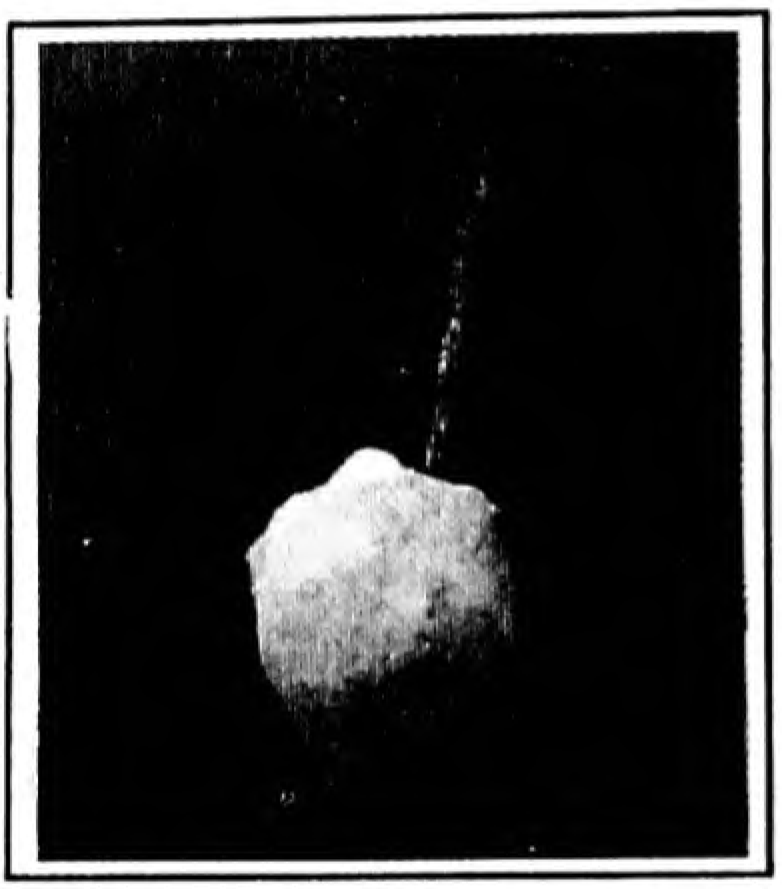
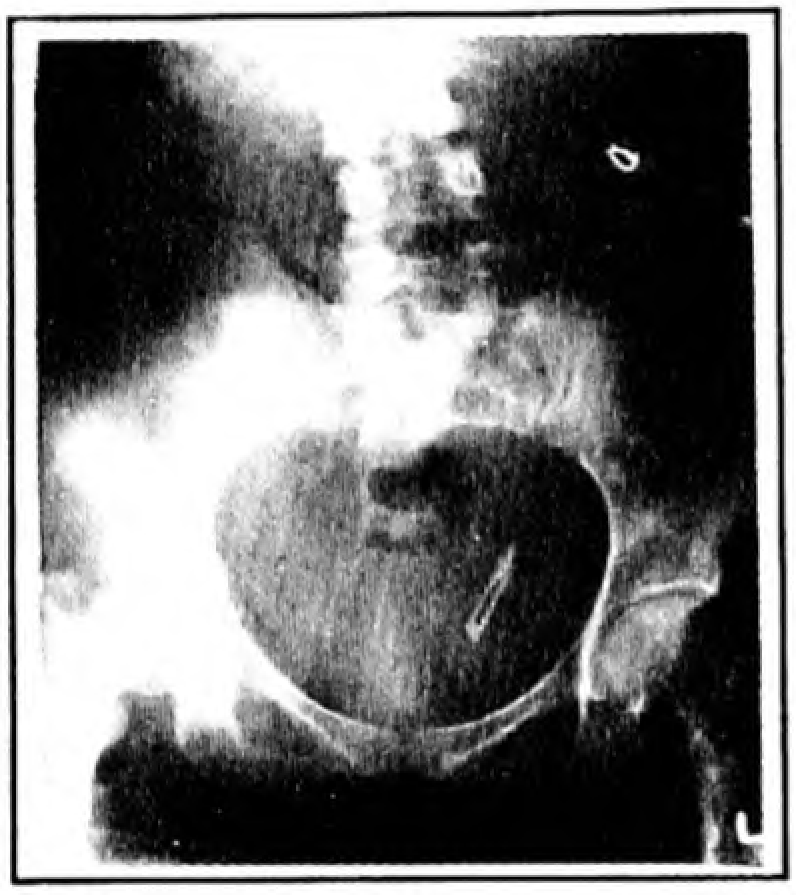
Introduction:

Foreign bodies of urinary bladder is not a rare entity. Most of the foreign bodies may be self inserted for sexual gratification or may be inserted for therapeutic purpose and neglected in complication or may be iatrogenic by a non vigilant surgeon or assistant. We present three case reports of different foreign bodies in bladder.



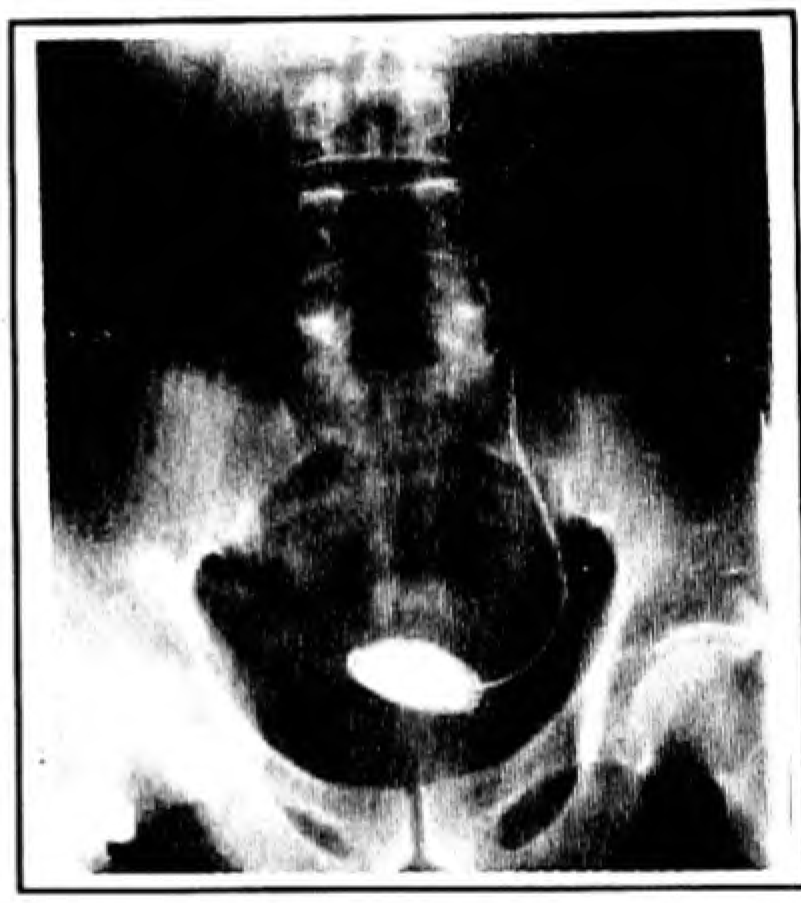


Case Report No. 1

Twenty years old female was seen in urology out patient department with chief compliants of burning micturation with frequency and urgency for last two months. Her routine urine microscopy showed puss cells of 80-90 hpf with 10-12 RBC hpf. X-ray of KUB region showed a stone with an elongated stalk in the bladder area. Ultra sound confirmed the X-ray finding. Cystopanendoscopy revealed a long wire like material with dense encrustations around it. Suprrapubic cystostomy was done and foreign body embed with stone was removed, after breaking the encrustations it turned to be a hair pin. On questioning the patient in post operative period it was found that she had introduced it for her sexual gratification.

Case Report No. 2

Twenty eight years male patient presented with gross haematuria with excruciating supra pubic pain. Patient gave history of cystolithotomy ten days back though no details of surgery were available with the patien. Clinically patient had a recent suprapubic scar which was tender. X-ray abdomen revealed a scalpal blade in suprabubic region. Emergency cystostomy was done and blade was removed from the bladder, patient had uneventful recovery.



Case Report No. 3

Thirty years old male patient presented with intermittent painful heamaturia and dysuria for last two motnhs. Patient gave history of left uretrorenoscopic extraction of left lower uretric calculus with stenting one year back. Routine investigaiton revealed puss cells 10-15hpf with RBC 20-30hpf. X-ray of KUB region showed a double J stent on left side with encrustations in its entire course with stone formation at its lower end. Patient gave history that he had lost to follow up with his operating surgeon due to his domestic reasons. Patient was prepared for cystoscopy and uretrorenoscopy but on cystoscopy the stent was gently

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pulled into the bladder. The stone at its lower end was crushed by doing cystolithopaxy. Stent was removed through cystoscope and stone fragments were removed by bladder washes.

Discussion

Foreign bodies in urinary bladder are not uncommon and many kinds of foreign boidies have been reported in the urinary bladder. Davidor reported a case of forty seven years old man who had self inflicted a piece of copper wire[1]. Tan et al had reported four cass of foreign bodies in urinary bladder[2]. Most of the patients with self induced foreign bodies are too ashamed to admit they had introduced it for sexual gratification or perversion and were diagnosed only when got into complication. known foreign bodies seen in the urinary bladder as in our case like hair pin, neglected stents, sometimes fractured foleys cather ballon, has been noticed to be the nidus for stone formation. Most of the foreign bodies in urinary bladder are diagnosed by X-ray pelvis or by ultrasound and sometimes picked up on cystoscopy. Gourment reported a foreign body in the urethra for erotic stimulation which was lost in bladder and after seven years presented with obstructive urinary symptoms[3].

In most cases foreign bodies are removed endoscopicaly which include neglected stents, small wire pieces or fractured foleys cather ballon, but sometime sharp objects like scalpal blade as in our case report needed open cystostomy as if attempted endoscopically could cause more damage to urethra and bladder. Keeping in mind the clinical presentation of our cases and on reviewing the literature it is mandatory to keep in mind the possibility of foreign body as differential diagnosis in patients presenting with chronic dysuria or cystitis or haematuria with or without history of surgical intervention not responding to chemotherapy.

References:

1. Davido MI. A case of multiplayer presence of foreign body in bladder. Urol. Nefrol (Mosk), 1997 July;4:40-41.
2. Tan LB, Chiang C, Huang CH, Chou YH and Wang CJ. Foreign body In the urinary bladder. Kao. Hsiung. I., Hsueh Tsa: chih 1993 Oct (10);604-609.
3. Rabii R, Bennani S, Rais H, Hafiani M, el Mirini M and Benjelloun S. Foreign bodies of bladder and urethra. Apropos of 2 cases Ann Urol (Paris), 1997;31(5):291-293.
4. Grumet GW. Pathologic masturbation with drastic consequences: case report J. Clin. psychiatry 1985 Dec;46(12):537-539.
5. Chamary VL. An unusual cause of iatrogenic bladder stone. Br. J. Urol. 1995 July; 76(1):138.

HEPATOPROTECTIVE ACTIVITY OF EMBELIA RIBES AGAINST PARACETAMOL INDUCED ACUTE HEPATOCELLULAR DAMAGE IN MICE

Nahid Tabassum; Shyam S. Agrawal

Abstract: *Embelia ribes commonly known as vidanga, has been reported to be useful in jaundice. It is a constituent of various formulations marketted for liver ailments. However, no systemic scientific study is available on its efficacy as a hepatoproctive agent. The proctective effect of Embelia ribes on paracetamol induced liver cell damage was studied using mice as experimental animals. Paracetamol was administered orally in a dose of 500mg / kg body wt 48hrs before the administration of drugs. The mice treated with Embelia ribes extract (50,100 & 200 mg / 100g/day) showed a dose dependent fall of 41%, 47%, & 66% respectively in the serum SGPT levels as compared to the elevated levels in the mice receiving paracetamol only. Histopathology of liver of mice revealed 67%, 70% and 80% normal livers respectively in mice receiving the above doses of E ribes. The results suggest that extract of E. ribes possesses hepatoprotective activity against paracetomol induced acute hepatocellular damage in mice.*

Key words: *Paracetamol, Embelia ribes, Hepatoprotective, Mice.*

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Introduction

Emeblia ribes Burm. (Vidanga, N. O Myrsinaceae) is a constituent of various formulations marketed for liver ailments. Fruits of Embelia ribes have been shown to posses analgesic, antipyretic (Atal & Sidique 1984)[1], antibacterial (Narang et al 1961)[2] antifertility (Seshadri et al 1981[3], Agarwal[4] et al 1986) and anthelminithic (Chopra[5] 1966) activity. It has also been reported to be useful in jaundice (Chopra 1966). However, no report in available on the efficacy of E. ribes as a hepatoprotective agent. So in the present study, E. ribes was screened for hepatoproctective activity against acute hepatotoxicity induced by paracetamol.

Material & Methods

Embelia ribes (ER) seeds were purchased from the local market in Delhi. After grinding to a coarse powder, 50% ethanolic extract was prepared by cold maceration as described in Indian Pharamacopoeia (1985)[6]. The dried mass of the extract was macerated with 1% gum acacia and suspended in distilled water to be given orally with a catheter (ER 50,100,200 mg / 100g).

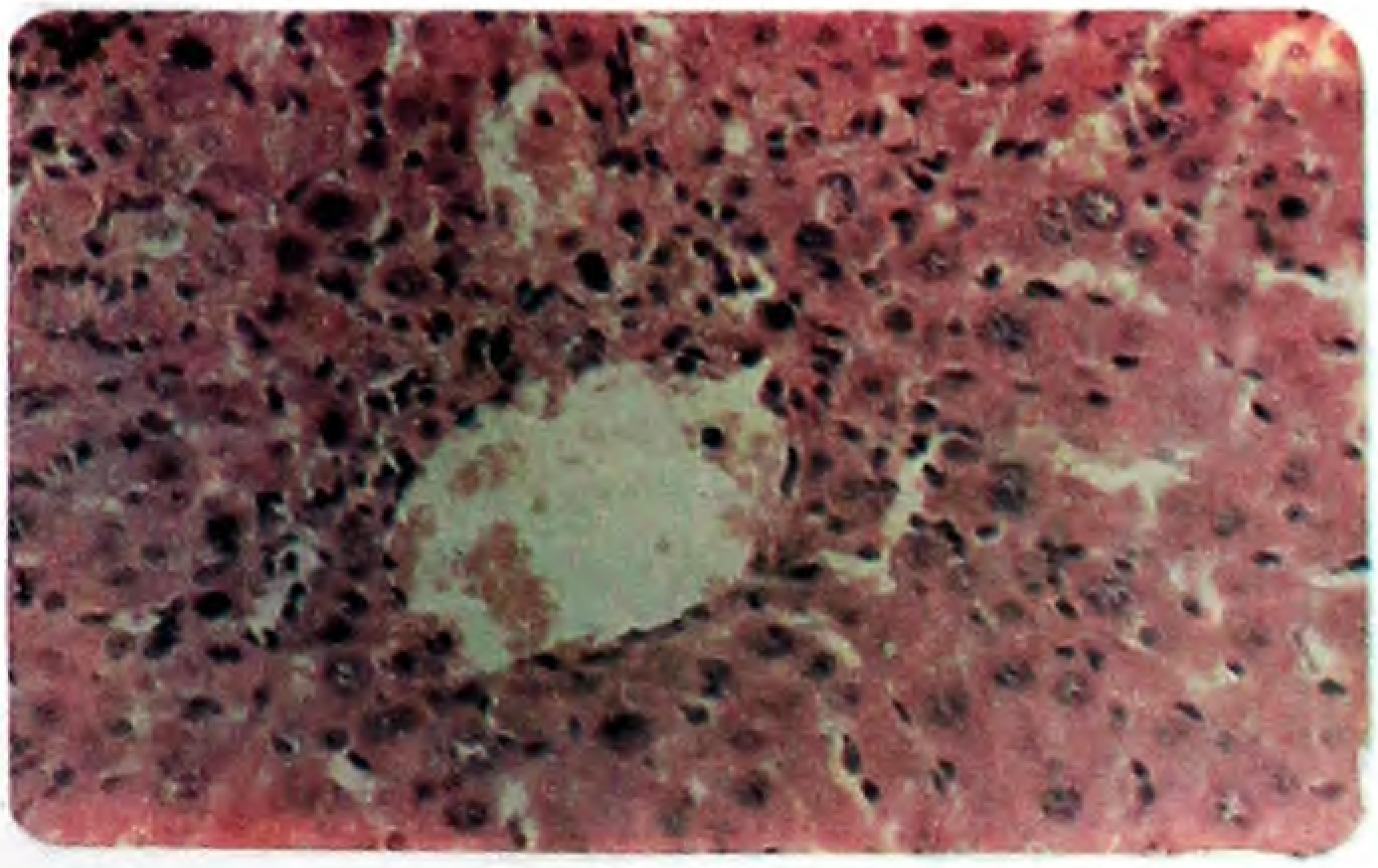

Fig.1: Centrizonal Necrosis after administration of single dose of paracetamol (500mg/kg) (H&E x 400)

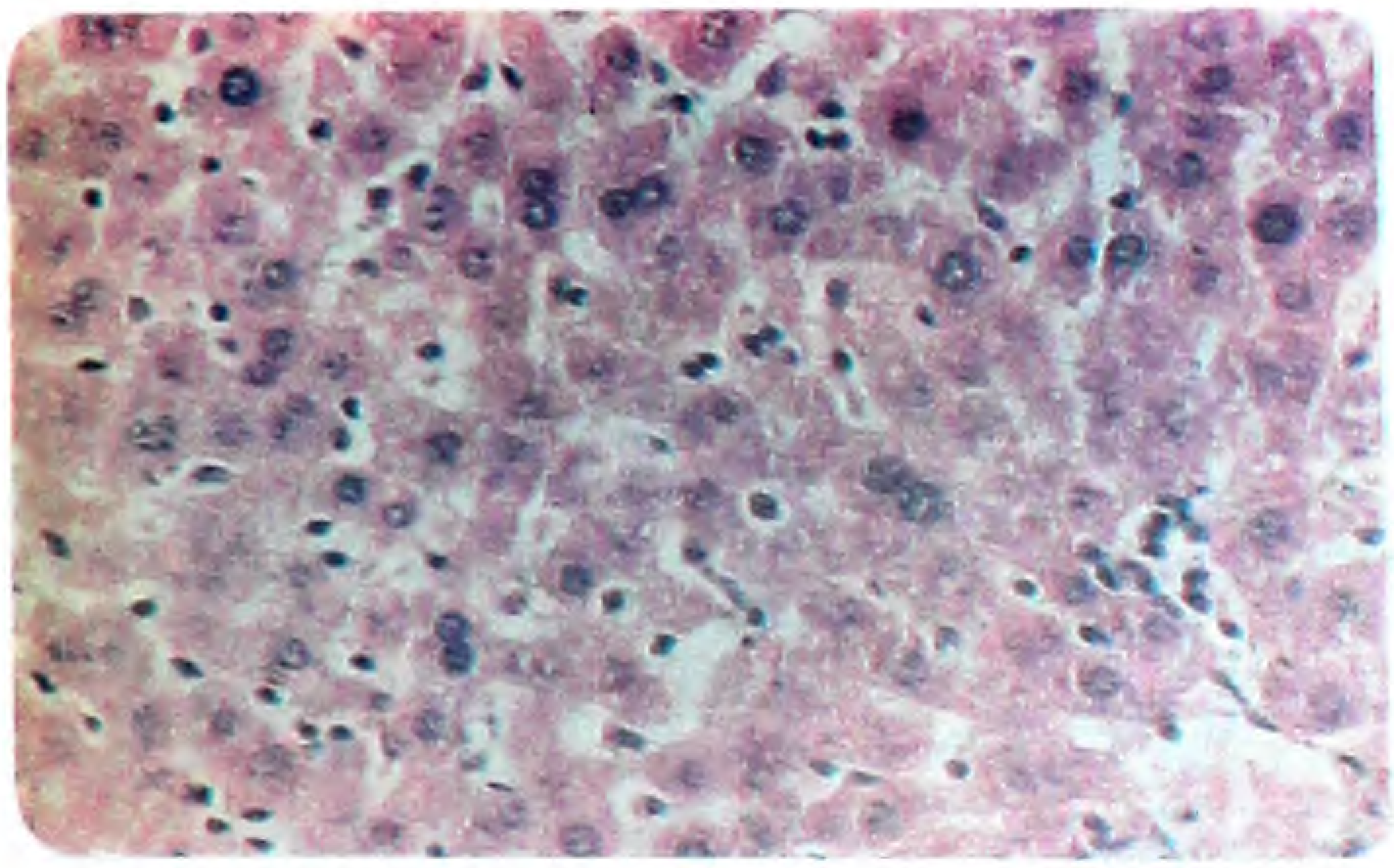

Fig.2: Anisonucleosis and binucleate cells in the liver treated with paracetamol (500mg/Kg) and E. ribes (100 mg/100g/day) (HxE x 100)

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Animals and exposure conditions

Healthy albino mice (15 to 30g) of either sex were housed under uniform husbandry conditons and given pelleted diet (Lipton's India limited) and water ad-libitum. The animals were housed at a temperature of 25+ 1 degree Celcius with a 12h light / dark cycle.

Paracetamol Induced Hepatocellular Damage

Mice were divided into five groups of 6 animals each. Group 1 served as normal control and received 1% gum acacia suspension only. All other groups II, III, IV received paracetamol once (500 mg/kg, po, aqueous solution) (Pandey & Shrivasrava, 1990)[7] with group II serving as paracetamol treated control. 48hr. after paracetamol adminsitration, Group III, IV & V received different doses (50, 100 & 200mg/100g/day) p.o, of ER extract once daily for five consecutive days.

16th day after the administration of last dose of drugs, animals were anaesthetised with ether & blood was collected from the retino-bulbar venous plexus & serum was separated by centrifugation for glutamic pyruvate transaminase (SGPT) levels (Reitman & Frankel,1957)[8] which has shown to be elevated by paracetamol (Dhawan et al, 1991). Mice were killed, livers excised, rinsed clean in saline and preserved in 10% formaline for histopathological study (using five um micro meter thick sections stained with haematoxylin & eosin, H&E). Results were statistically analysed using students t' test.

Results

Biochemical studies revealed a dose dependent fall of 41% ($p<0.05$); 47% ($p<0.05$) & 66% ($p<0.01$) in the SGOT levels respectively, after the administration of 50mg (122.6-±6.34 IU/L), 100mg (116.70 ± 14.13 IU/L) and 200mg (97.25 + 2.79 IU/L) of ER extract as compared to the elevated SGPT levels (163.72 IU/L) in the mice receiving paracetamol only.

Histopathalogical examination revealed portal and parenchymal inflammation (3+) and marked centrizonal necrosis in 38% livers that received only paracetamol (Fig 1). 50% ethanolic extract of E. ribes at the dose of 50mg/100g/day showed 70% of livers as normal while portal inflammation was seen in 30% livers. Kuffer cell hyperplasia was observed in one of the livers inthe group treated with 100mg/100g/day of E ribes extract (Fig. 2). E.R. treated mice (200mg/100g/day) revealed portal inflammation (1+) in 20% and parenchymal inflammation 91+) in 40% of mice. The mice administered E. ribes extract of different doses also revealed the presence of binucleate cells and anisonucleosis showing regeneration of liver cells.

Discussion:

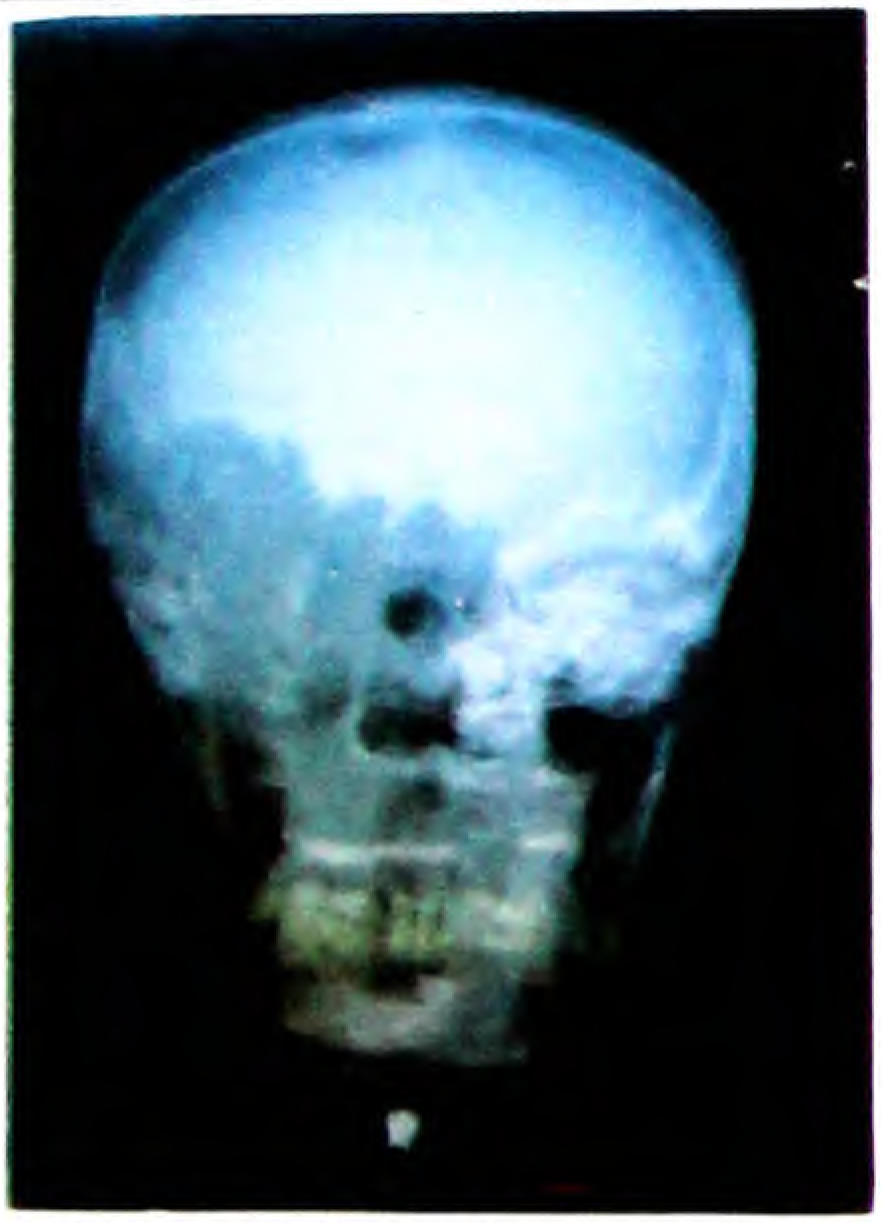
Paracetamol causes acute centilobular hepatic necrosis in rots, mice, guinea-pigs, hamsters rabbits, cats dogs and pigs[10] (Boyd and Bereczkey, 1996)[10]. Mice and hamster are very sensitive while rat is very resistant[11]. So, in the present study mice were used for producing hepatotoxicity with paracetamol. Only serum glutamate pyruvate transminase (SGPT) levels were estimated as it is the more specific index of liver cell damage.[12] Centrizonal haemorragic necrosis is the characteristic lesion in humans[13] & in experimental animals[14]. In the present study, ER extract (50, 100 & 200mg / 100g/day) produced significant folls of 41, 47 & 66% respectively in SGPT levels in mice as compared to elevated levels in the group receiving only paracetamol indicating that the degree of hepatic cell damage was of lesser magnitiude in ER treated mice (Fig. 2,3,4). Histopathological studies of liver confirmed the attributivity of the observed alterations of SGPT levels to hepatic damage on health. The study has shown that livers challenged with paracetamol have centrizonal necrosis and parenchymal inflammation. In animals administered with above doses of ER, there was no noticeable hepatocellular necrosis. Only mild parenchymal inflammation was observed while binucleate cells and anisonucleosis was seen in these mice which are an indication of hepatic cell regeneration.

These observations suggest that extract of Embelia ribes possesses hepatoprotective activity against paracetamol induced acute hepatoceullular damage in mice.

References:

1. Atal C.K, Siddique MA; Zutshi V.A.; Non-narcotic, orally effective, centrally acting analgesic from an ayurvedic drug. J. Ethno Pharmacol. 1984;11(3):309-317.
2. Narang G.D, Garg L.C & Mehta R.C.: Preliminary studies on antibacterial activity of E. ribes (myrisinaceae). J. Vet. Animal Husbandry. Res 1961;5(1):73-79.
3. Seshadri C & Vekataraghavan S: effect of aqueus and alcoholic extract of E. ribes on liver plasma and reproductive organs in male rats, a preliminary study Indian J., Pharamacology. 1981:13(1):88.
4. Agrawal SS, Garg A & Agarwal S: Screening of Phyllanthus niruri Linn. and Ricinus communis Linn. on alcohol induced liver cell damage in non-hepatomized and partially hepatectomized rats. Indian. J. Pharmacology 1986;18:P211-214.
5. Chopra R N, Nayar SL, Chopra IC: In; Glossary of Indian Medicine, CSIR Publ. New Delhi 1966;104.
6. Pharmacopoeia of India, official Publication. edition 1985.
7. Pandey GP & Srivastava DN: Effect of Livol and some of its ingredietns on paracetamol idnuced hepatotoxicity in mice. Ind. J. Pharamc. 1990:2291):12-13.
8. Reitman S & Frankel S: A colorimetric method for the determinanation of serum glutamatic oxaloacetic and glutamic pyruvic transaminase. Am. J. Clin. Path. 1957;28:56.
9. Dhawan BN & Dubey MP: Screening of plant for Biological activity. Part I. Ind. J. Exp. Biol. 1980;18(6):594-606.
10. Boyd E M, Bereczky G M: Liver necrosis from paracetamol. Brit. J. Chemother. 1966;26:606-614.
11. Jollow DJ, Thoregeirsson SS: Acetaminophen induced hepatic necrosis VI. Metabolic deposition of toxic and non-toxic doses of Acetaminophen. Pharmacology 1974;12:251-271.
12. Black M: Acetaminophen Hepatoxicity. Gastroent. 1980;78:382-392.
13. Clark R, Thompson R.P.H., Bori rackanyaval V, Windoop et al: Hepatic damage and death from overdose of paracetamol. Lancet 1973;1:66-69.
14. Mitchell J.R, Thorgeirsson S.S: Accetaminophen induced hepatic injury Protective role of glutathione in man and rationale for therapy. Clin. Pharmacol and Therap. 1974;16:676-684.

AN UNUSUAL BILATERAL CANINE IMPACTION IN MANDIBULAR SYMPHYSIS



A 30 year old lady come with complaints of a discoloured swelling in the lower anterior region, without any pain for last few months. On examination a swelling in the labial vestibule in relation to the lower incisors was found with following characteristics.

- Well circumscribed, localized, swelling,
- Bluish coloured giving a look of haemangioma,
- Non fluctuant, no pulsations,
- Firm to touch,
- Overlying mucosa non inflammatory. Freely moving over the underlying swellinigi suspecting a bony cystic lesion.

There was no local lymphadenopathy.

1. Radiography revealed periapical and occlusal two horizontally impacted canines (left coronal to the right one) found near the apices of the lower incisors on the labial side with an associated radolucency of the size of about 1.5 cm in diameter. (well depicted in the radiograph)
2. *FNAC of the suspected lesion:* The patent was operated under L.A. After disinfecting the area with betadine wash, an incision was given along with gingival margin from first premolar to premolar with two releasing incisions. (Trapezoid flap). The trapezoid flap raised by blunt disection along with the underlying periosteum. Thin shell of bone overlying the cyst removed by small sharp chisel and thus epithelial lining was freed from the bone and the underlying cyst enucleated in toto. The surrounding bone was chiseled in such a way to first laxate the tooth and then extract it out. After extracting the left impacted canine, right impacted canine approached by chiselling the surrounding bone. The tooth gradually got exposed and laxated and finally extracted out. The area flushed with normal saline. Remaining bony chips removed. sharp bony margins smoothened. The area again flushed with normal saline. The margins of the cavities reduced with bone roaengeurs in order to sausarize it so that overlying mucosal flap and the underlying bone will come into intimate contact so as to avoid any haematoma formation. The area again flushed with normal saline. The flap sutured back to its palce by interrupted sutures with silk (3-0).

   The patient advised to hold the pressure pack for an hour and half & usual post operative instructions given:-

Discussions

A tooth is said to be impacted when its formation is complete yet has not erupted to its final position to assume its normal anatomical arch relationship even beyond its chronological eruption date. In the order of rate of frequency of impacted teeth, mandibular canines rank No. 5. In other words mandibular canines are less frequently impacted as compared with third molars, maxillary canine and mandibular premolars.

Regarding the etiology of the impaction of mandibular canines, besides systemic and other local factors, the important etiological factors responsible are:-

1. Longer distances to travel.
2. Change in the pattern (normally permanent canines lie lingual to the primary one) leading to premature loss of the preceding deciduous tooth.
3. Delayed resorption of primary canines.
4. Eruption between the teeth already erupted (mesiodistal daimeter of primary canine is less then permanent canine).

Systemic conditions to be taken into consideration are:-

1. Hereditary cause.
2. Condition like rickets, anemia, T.B., malnutritioin, endocrnal dysfunction, congenital syphilis etc. which cause interfere in the normal development of a child.
3. Skeleton Disorders like, cleido-cranial dysostosis, oxycephalis, achondroplasia etc. causing skeletal deformities.
4. Cleft lip/plate.

As the classification of mandibular canine is concerned these are classified as vertical/ horizontal / oblique.

Occasionally beneath the apices of the central incsors. Unusual positon like lower border of mandible mental protuberance, apposite side crossing midline.

Indications for extraction depend on:-

i. Pressure causing change in the positon of the adjacent teeth.
ii. Cyst formation.
iii. In case of endutulous arch, pain during denture wearing.
iv. Pain radiated to other areas.
v. May start erupting in an unusual poistion.

As the surgical management of the impacted mandibular canines in a young patient is concerned there be a combined effort of the surgeon and orthodontist so that a chance may be provided to it for eruption at the normal position.

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JK-Practitioner 2002;10(1): 45-46

NON-ANEMIC IRON DEFICIENCY - DIAGNOSTIC PERSPECTIVE:

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Anemia is known to mankind from times immemorial Mummy of an Egyptian queen in Cairo museum who died of anemia to the story of Iphyclus in Greek mythology, of cure of impotence, by drinking iron rust, dissolved in wine are pointers towards that use of Loha Bhasam for treatment of anemias has been known to Ayurveda for thousands of years. The Unani and Chinese system of medicine do possess references about anemia[1].

Nutritional anemia is an important problem affecting large population groups. Basically the deficiency states of hemopietic nutrients such as iron, B12, folic aid and protein all lead to anemia. Anemia itself contstitutes about 65% of hematological disorders seeking hospital admission.

WHO expert group in 1970 found that in Asia approximately 10% of men, more than 20% of women, 40% of those in pregnancy, more than 50% children of all ages and 92% of children below the age of 2 years suffer from iron lack anemia. WHO technical series criteria for diagnosis of anemia being Hb < 10.5 gm/dl, PBF hypochromia and MCHC _ 31%[2].

However, iron deficiency without anemia is an entity or symptom complex deserving mention and attention. This symptom complex can cause fatigue, affect attention span, alertness and learning. Anorexia, depression, palpitations and vertigo in non-anemic subjects are some other accompaniments[3-5].

Walderistrom (1938) reported that dysphagia a concomitant of iron lack anemia could occur without anemia and was equally susceptible to iron therapy. This condition of iron deficiency without anemia was labelled as sideropenia.6

Heinrich (1968) described iron deficiency without anemia as most common deficiency in man. He described it to manifest in two stages "Preiatent" and "Latent" iron deficiency. Perelatent iron deficiency is charactrised by depleted iron stores, increased iron absorption, with normal serum iron and iron binding capacity. Latent iron deficiency was described as more advanced stage of iron depletion with low serum iron levels. Increased total iron binding capacity but without anemia.[7]

Even as now iron deficiency, both overt and covert, is being diagnosed in laboratory by estimating hemoglobin concentration, PBF, MCV, serum iron, iron binding capacity, percent transferrin saturation and bone marrow smear examination for iron staining. All these parameters have limitations in children. MCV is variable with age. microcytosis is an intrinsic feature of erythropoiesis. Technique used for estimation of serum iron and iron binding capacity have their limitations and further require volume of blood, usually difficult to obtain in pediatric age group. Transferrin saturation percent, though relatively reliable, yet large biological variation in serum iron can result in false positive and negative diagnosis.[5-5]

Free erythrocyte protoporphyrin (FEP) estimation is useful for early detection of iron deficiency. It also distinguishes thallassemia from iron deficiency but FEP does vary due to developmental changes in R.B.C.'s and is alo evevated in inflammatory diseases and on exposure to lead.[4]

Absence of iron on bone marrow examination can be used as a criterion for iron deficiency without anemia, because it is a direct measure of storage content. Stainable bone marrow is graded as 0-6.[8]

0	No visible particles	0.1	(oil immersion)
I	Iron particles visible	0.1	
II	Sparsely disturbed particles	H.P.	(High power)
III	Numerous small iron particles	H.P - L.P.	
IV	Large particle aggregation	L.P.	(Low power)
V	Large clumps	L.P.	(Naked eye)
Vi	Very large deposits intra and Extra-ceullular (Naked eye)		

Though bone marrow examination is nothing more than an intraosseous infection, yet it has attendent reservations and therefore cannot be employed for mass screeining purposes to pick up iron deficiency without anemia.

An immunoradiometric method for determining serum ferritin is a relatively recent method for diagnosing latent iron deficiency and iron lack anemia. Serum ferritin is a high molecular weight compound, consisting of protein shell and hydrated ferric phosphate. Iron contitutes about 20% of compound, ferritin being a storage protein is found mainly in RE cells, liver cells and evolving red cell is coupled to the tube in excess contrary to RIA. using coated tubes. After reaction with antigen and washing125$_{I}$ labelled antibody is added to the reaction vessel and incubated second time. Antigen to be determined is then bound

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fromboth sides by two antibody molecules in a type of sandwich. The excess tracer is aspirated off and remaining radioactivity in the tube is measured. The method allows a simple separation of bound and free radioactivity. An advantage of this method over classic RIA is that directly after the first incubation serum and other possible interfering substances present are removed from reaction mixture.[9]

In order to establish the utility of serum ferritin assay in non-anemic children, a study was conducted at Children Hospital Srinagar in which eighty children, apparently healthy and non-anemic were chosen from pediatric follow up clinics. None had suffered from any serious medical or surgical illness and it was ensured that iron oral or parenteral was not consumed by them during preceeding three months. Another group comprised of twenty anemic children and anemia being of nutritional origin. A detailed history and clinical examination with emphasis on dietary history and anthropometry was carried out. Complete Heamogram, serum iron and iron binding capacity was also done.

It was found that mean serum ferritin levels were 31.4 ngms/ml in non-anemic children was found to be diagnostic of latent iron deficiency. Incidence of latent iron deficiency in non-anemic children was 26.8%. Besides, the sequential hematological response it was observed that clinical improvement in symptom complex still is the best and cheapest diagnostic tool to diagnose and monitor response to iron replacement therapy1 (Unpublished data)

References:

1. Mir MS : Non-anemic iron deficiency. (Thesis accepted by Kashmir University for MD degree (Pediatrics-1992)
2. Tripathi AM and Bhatia Nutritional Anemia in childhood. Ind. Pediatrics 12:343-346; 1975
3. Fielding et al: Iron deficiency without anemia, Lancet 3ii 9-10; 1965.
4. Nathan and Oski Hematology of infancy and childhood. Confirmatory tests for iron deficiency (3rd edition); 1981.
5. Wintrobe MM Iron deficiency and ir iron deficiency anemias. A text book of Cl hematology (6th edition); 1981.
6. Waldenstrom : Iron deficiency without anemia: Acta. Med. Scan 90:380; 1938.
7. Verlop: Iron depletion without anemia. Blood 36 (5) : 657-668; 1970.
8. Prakash et al : Significance of graded stainable marrow and erythropoiesis in hypochromic anemia in children. Ind. Pediatrics; 8:208-11; 1971
9. Addison GM et al: An immunoradiometric assay for ferritin in serum of normal subjects and patients with iron deficiency. J. Clin. Path 25; 326-329; 1972.

FINE NEEDLE ASPIRATION CYTOLOGY OF EXTRANODAL LYMPHOMAS - A STUDY OF 77 CASES.

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ABSTRACT: A retrospective review of FNAC over 11½ year period (January 1991 to July 2002) revealed 77 cases of extranodal lymphomas. These lymphomas were from various sites including soft tissue (24 cases), salivary glands (4 cases), oral cavity (3 cases), thyroid (14 cases), testis (2 cases), ovary (1 case) and bone (6 case). Nine cases were seen below the age of 14. Out of the remaining 68 cases, most i.e. 74% (57/68) were seen at the age of 30 and above with a wide age range of 30 to 90 years. There was an overall male predominance (48/77) except in breast and thyroid which together contributed more than half the cases (16/29) in females. The Working Formulation modified (to accommodate entities like marginal zone (MALT), mantle cell, lymphocyte/histocyte-rich B cell lymphoma) was applied to both FNAC and histopathology for comparison. Thus, on FNAC the diagnosis of 77 cases was: lymphoblastic lymphoma (8 cases), small lymphocytic lymphoma (3 cases), lympho-plasma-cytoma (1 case), plasmacytoma (1 case), small cell lymphoma (1 case), marginal zone lymphoma (1 case), large cell lymphoma (55 cases), Burkitt's lymphoma (2 cases), sarcoma NOS (3 cases) and seminoma (1 case). Histopathology comparison was available in 59 cases including 6 cases of lymphoblastic lymphoma, 3 cases of small lymphocytic lymphoma, 1 case of plasmacytoma, 45 cases of large cell lymphoma, 3 cases of sarcoma NOS and the case of seminoma testis. Cyto-histopathologic comparison was excellent in 10 cases including 6 cases of lymphoblastic lymphoma, 3 cases of small lymphocytic lymphoma and one case of plasmacytoma with 100% sensitivity and specificity. Out of 45 case of large cell lymphoma, histology showed large cell lymphoma in 43 cases, follicular and diffuse large cell in one case and marginal zone (MALT) lymphoma with transformation to large cell in one case. The 3 cases of sarcoma NOS on histopathology proved to be large cell lymphoma with sclerosis in 2 cases and non-cleaved cell lymphoma in one case. The case of seminoma testis also proved to be large cell lymphoma on histology. Thus, within the large cell lymphoma group totalling 47, in overall there were 4 misdiagnoses, giving an overall sensitivity of 93.5% and specificity of 100%.

The extra-nodal lymphomas in children are high grade, which are unlikely to be missed on FNA and deep - seated lymphomascan be managed without open biopsy. In adults, spectrum of extra-nodal lymphomas is more varied and the large cell lymphoma constituted the largest category. In conclusion, FNAC diagnosis of extranodal lymphomas in experienced hands can be rapid, reliable and cheap diagnostic modality without ancillary studies in majority of the cases.

***Key words:** Fine needle aspiration, Non Hodgkin Lymphoma (NHL)*

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INTRODUCTION

Lymph node aspiration was conducted by Greig and Gray(1) in 1904 in the USA. After 1950's it gained popularity in European countries. The publications from Radumhemmet of the Karolinska Hospital in Stockholm, Sweden(2,3) caught attention of pathologists world over. After late 1980s the role of FNAC has been well documented for the diagnosis of nodal lymphomas in several publications(4-9)

Extranodal lymphomas account for 26% to 40% of lymphomas.(10,11) Compared to nodal, the literature on the FNAC of extranodal lymphomas is limited(12-19).

Authors review their experience on FNAC diagnosis of extra-nodal lymphomas over last 11 1/2 years (January 1991 to July 2002).

MATERIAL AND METHODS

Eleven year period (January 1991 to December 2001) review on FNAC revealed 345 nodal (255 NHL and 90 HL) and 68 cases of extra-nodal non-Hodgkin's lymphomas. Added to the 68 cases are another 9 extra-nodal lymphomas encountered in 2002 (January to

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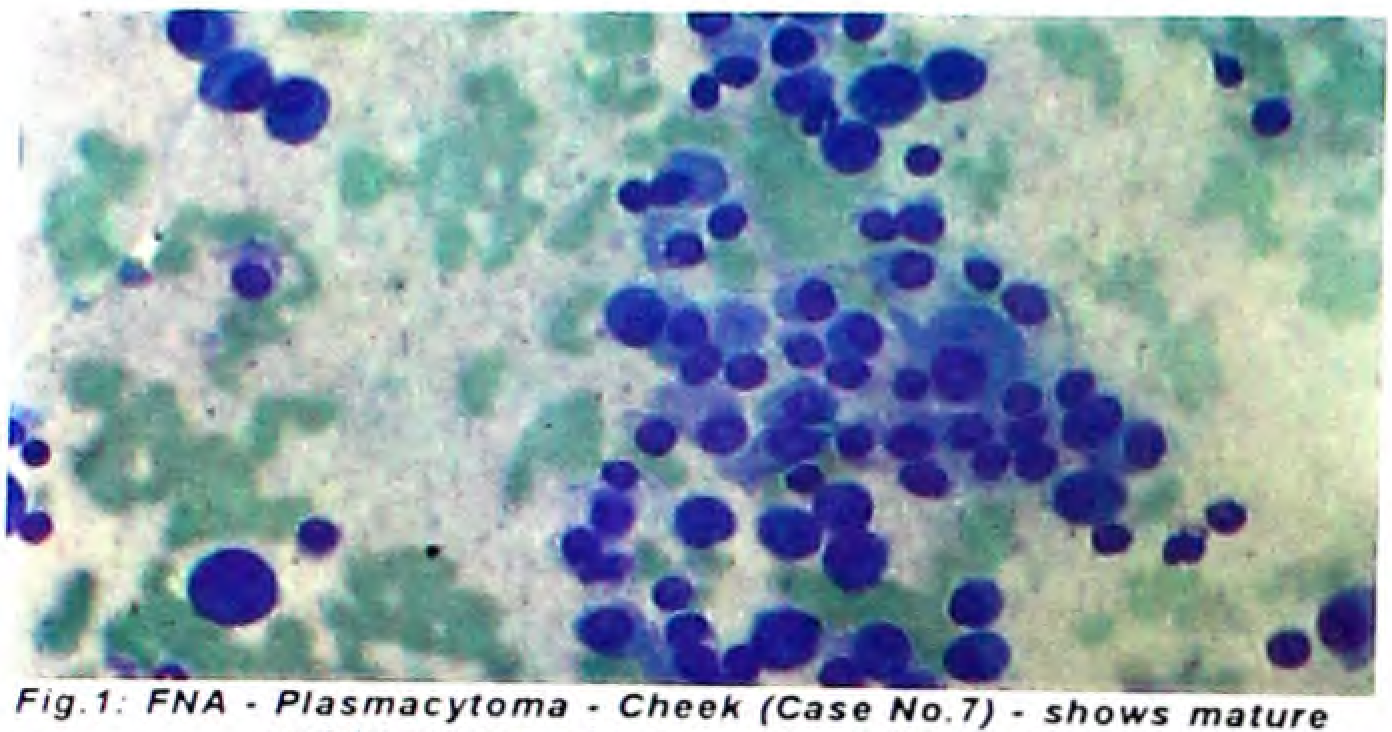

Fig.1: FNA - Plasmacytoma - Cheek (Case No.7) - shows mature and immature plasma cells. MGG x 400.

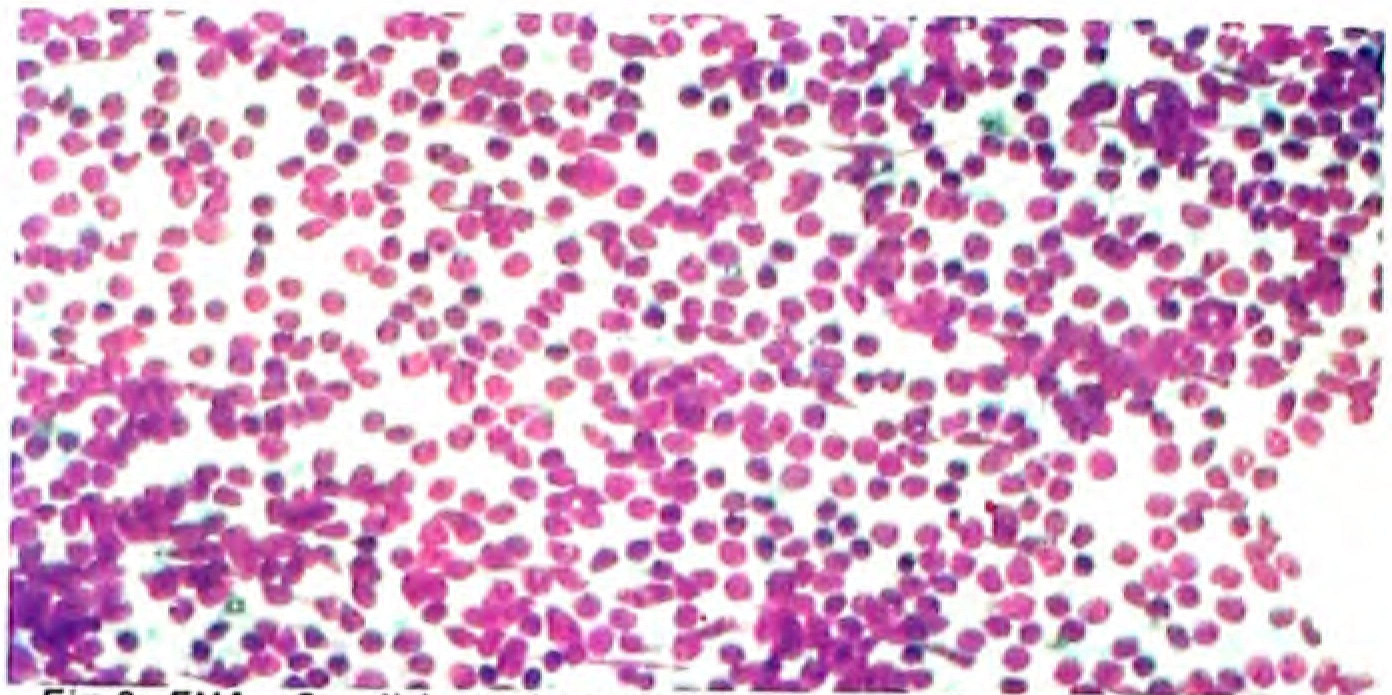

Fig.2: FNA - Small Lymphocytic lymphoma - Tarsal plate B/L (Case No.8) - shows diffuse sheets of small lymphocytes. MGG x 400.

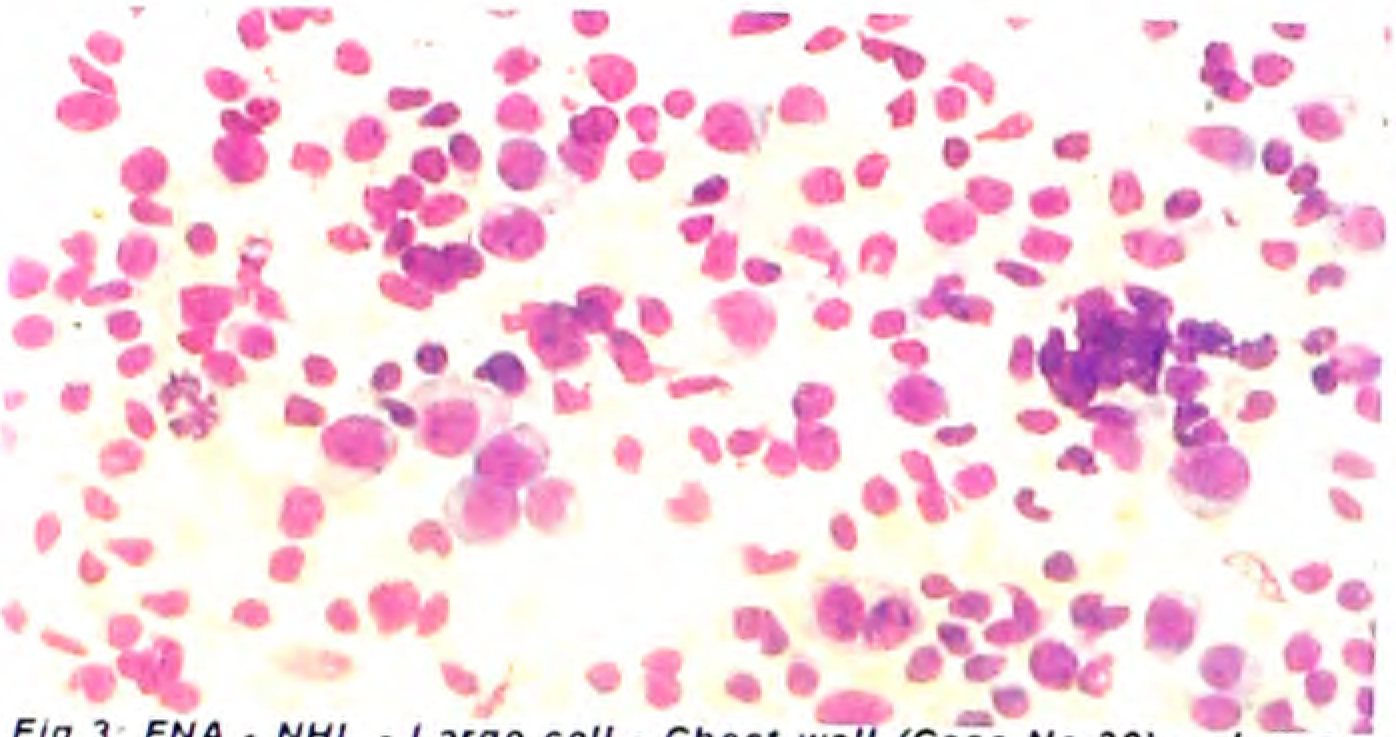

Fig.3: FNA - NHL - Large cell - Chest wall (Case No.20) - shows predominantly large cells and small Lymphoid cells MGGx400.

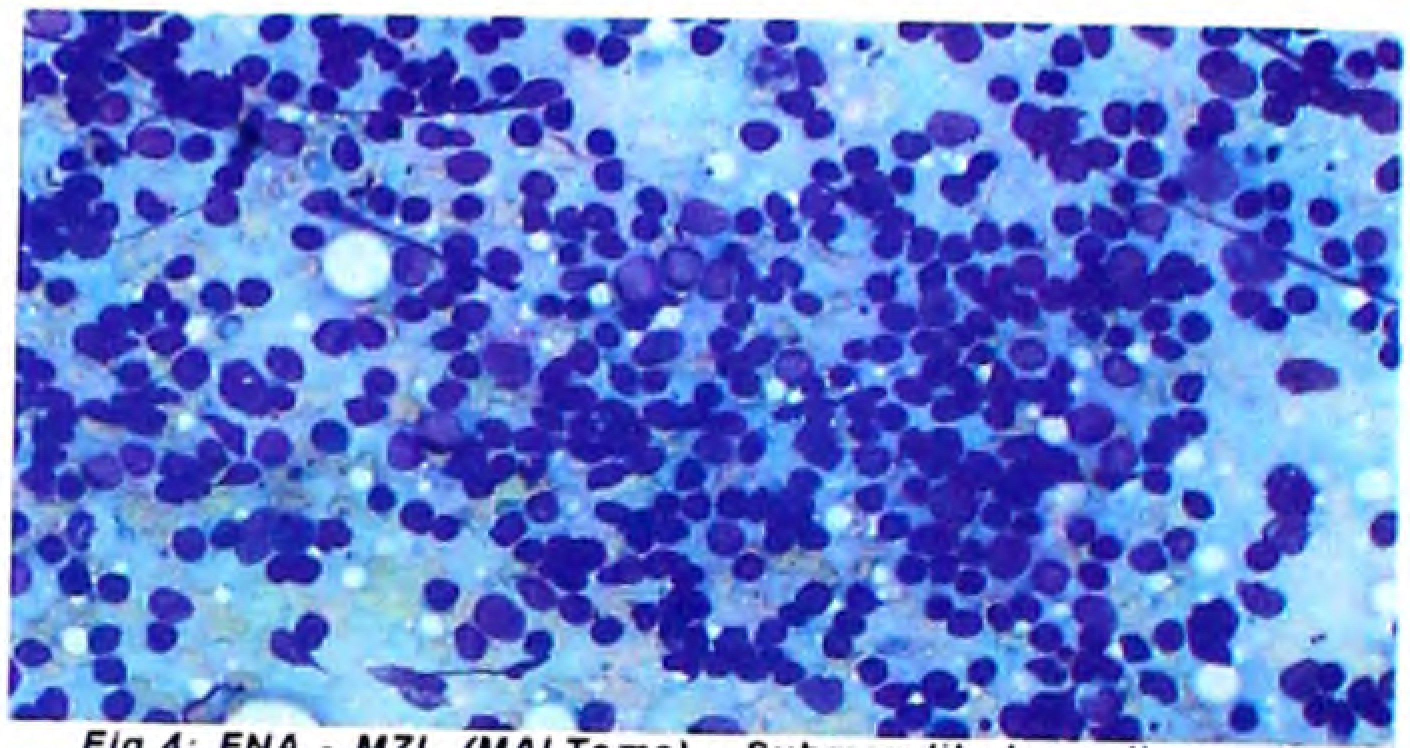

Fig.4: FNA - MZL (MALToma) - Submandibular salivary gland (Case No. 29) - shows medium sized and small Lymphoid cells MGG x 400.

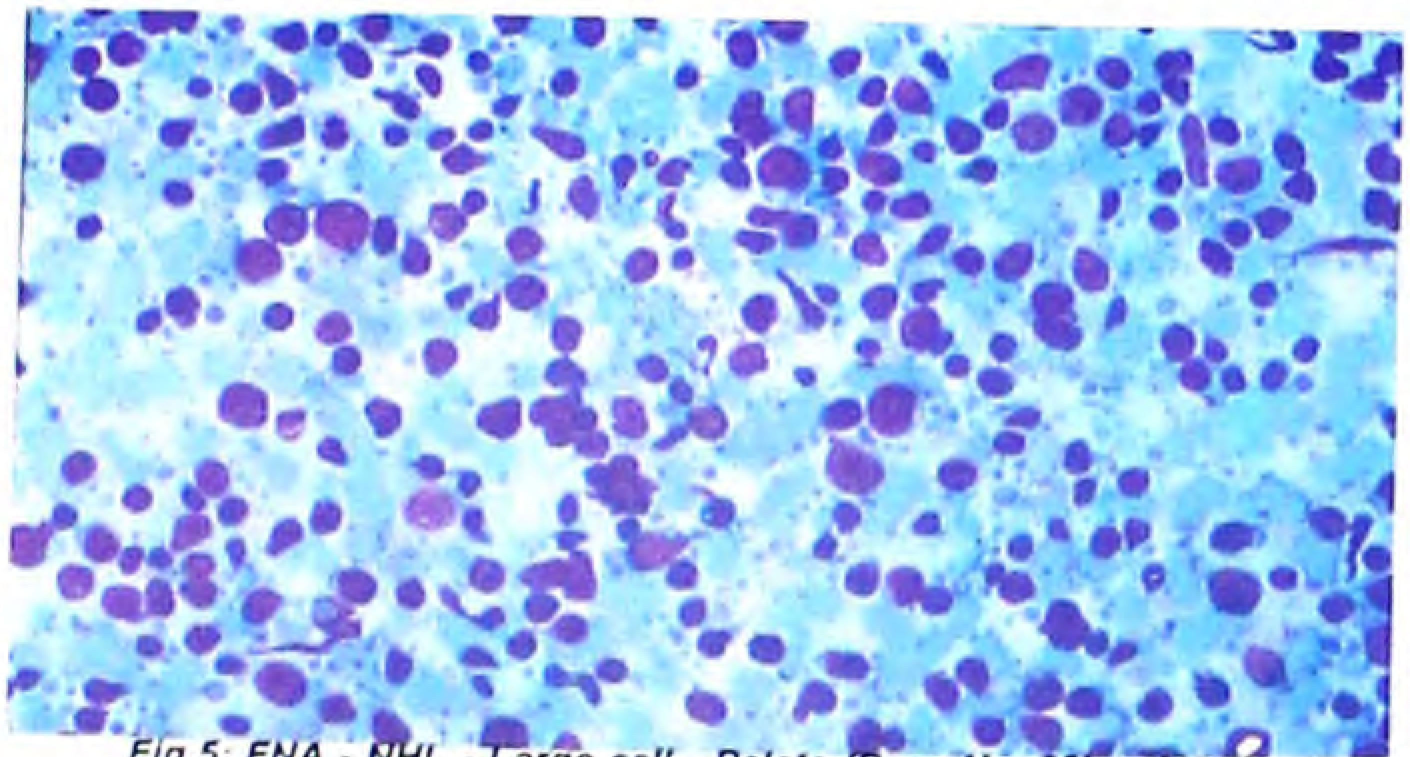

Fig.5: FNA - NHL - Large cell - Palate (Case No. 29) - shows predominantly large lymphoid cells with many lymphoglandualr bodies MGG x 400.

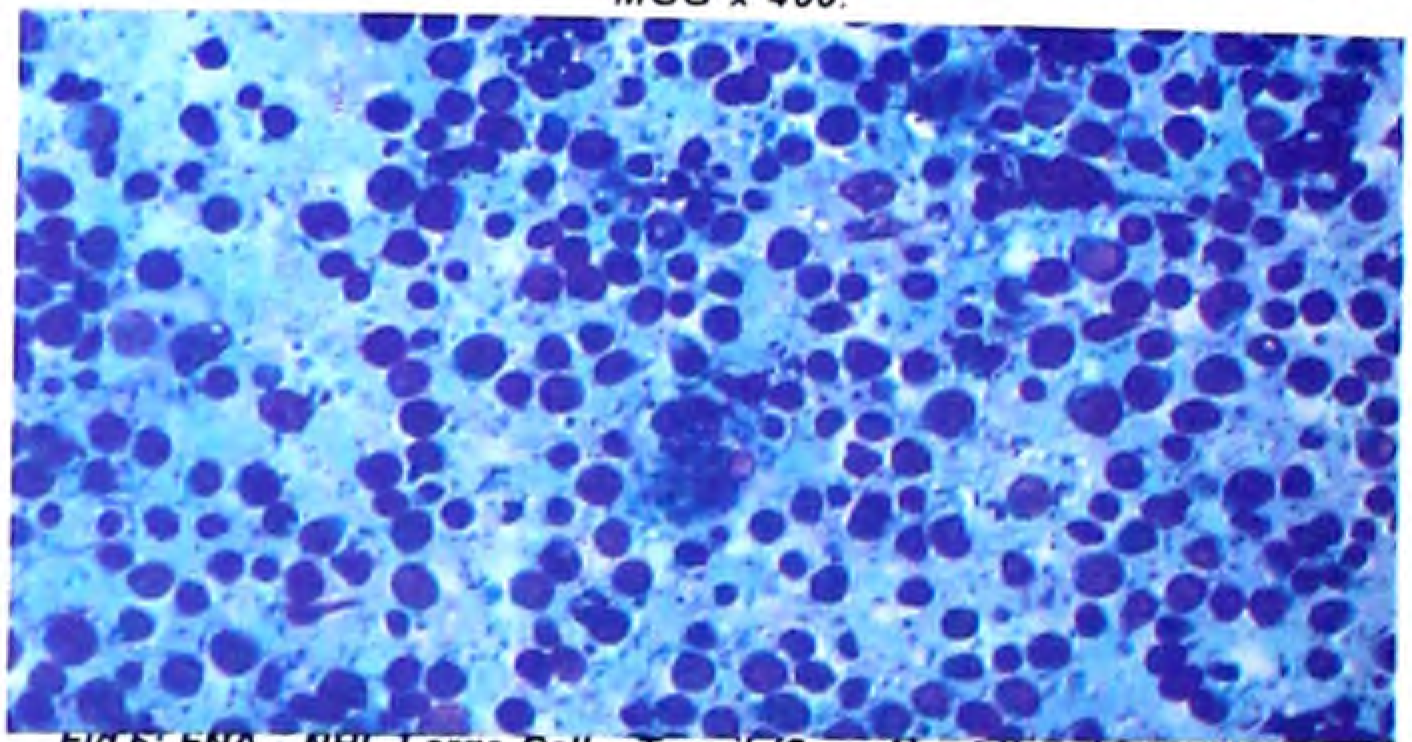

Fig.6: FNA - NHL Large Cell - Tonsil (Case No. 31) - shows dispersed large non-cleaved cells and many lymphoglandular bodies MGG x 400.

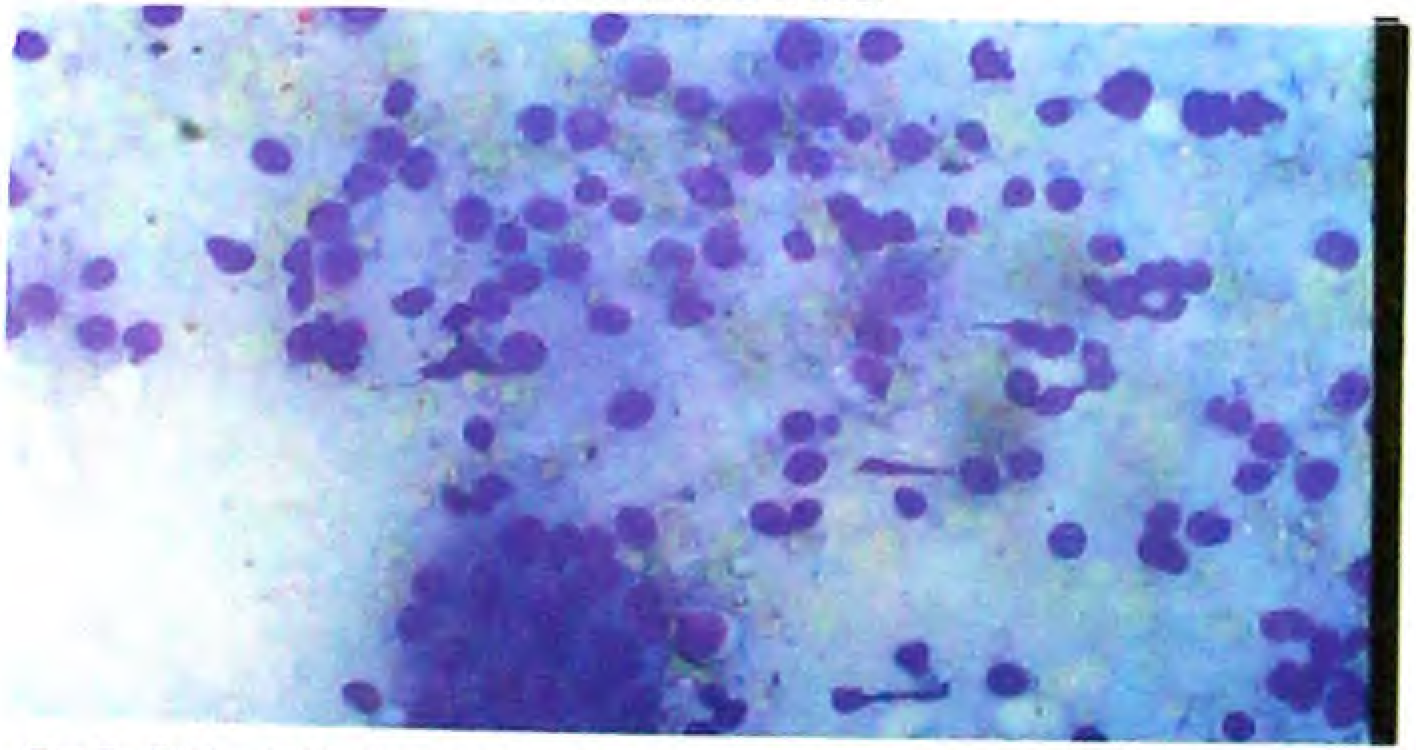

Fig.7: FNA - NHL low grade MZL (MALToma) - Thyroid (Case No. 35) - shows a follicle and dispersed medium sized lymphoid cells MGG x 400

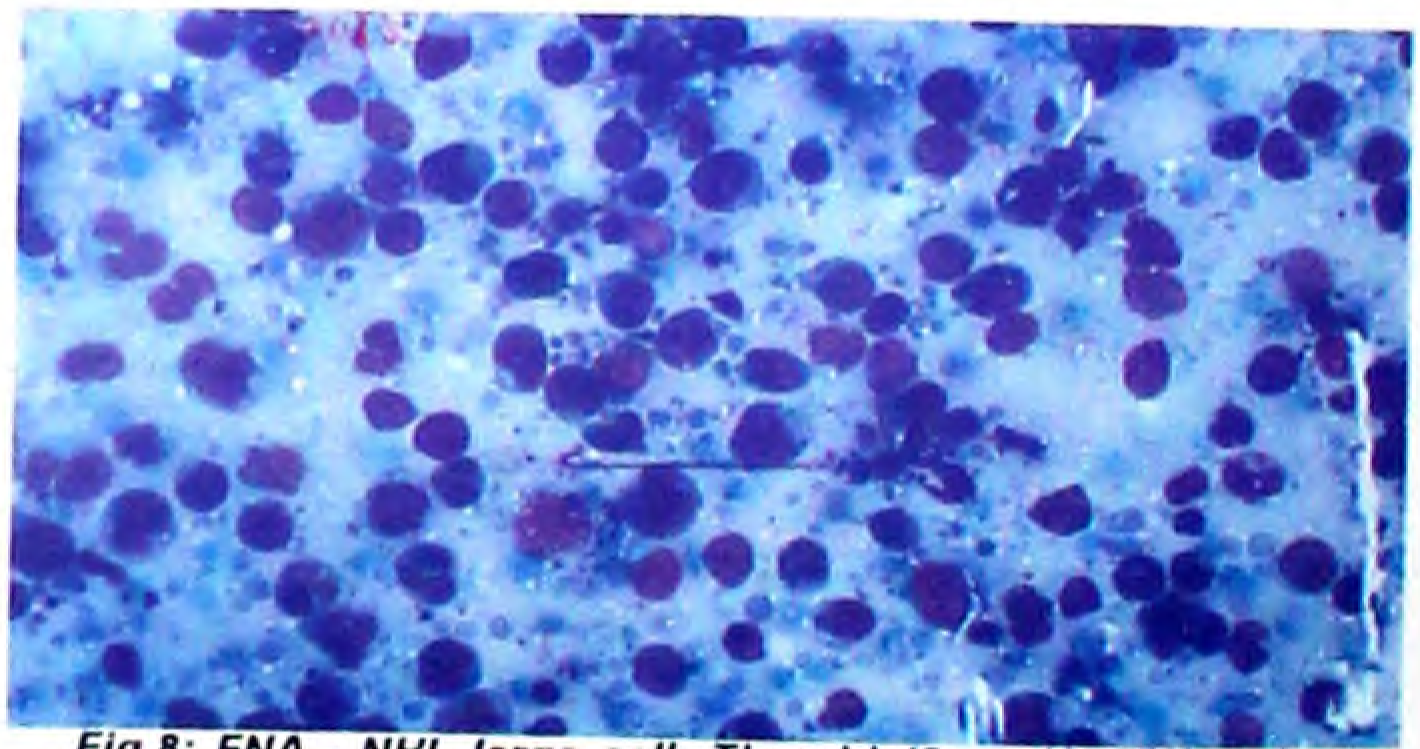

Fig.8: FNA - NHL large cell- Thyroid (Case No. 50) - shows large lymphoid cells some with lobated nuclei and many lymphoglandular bodies MGG x 400.

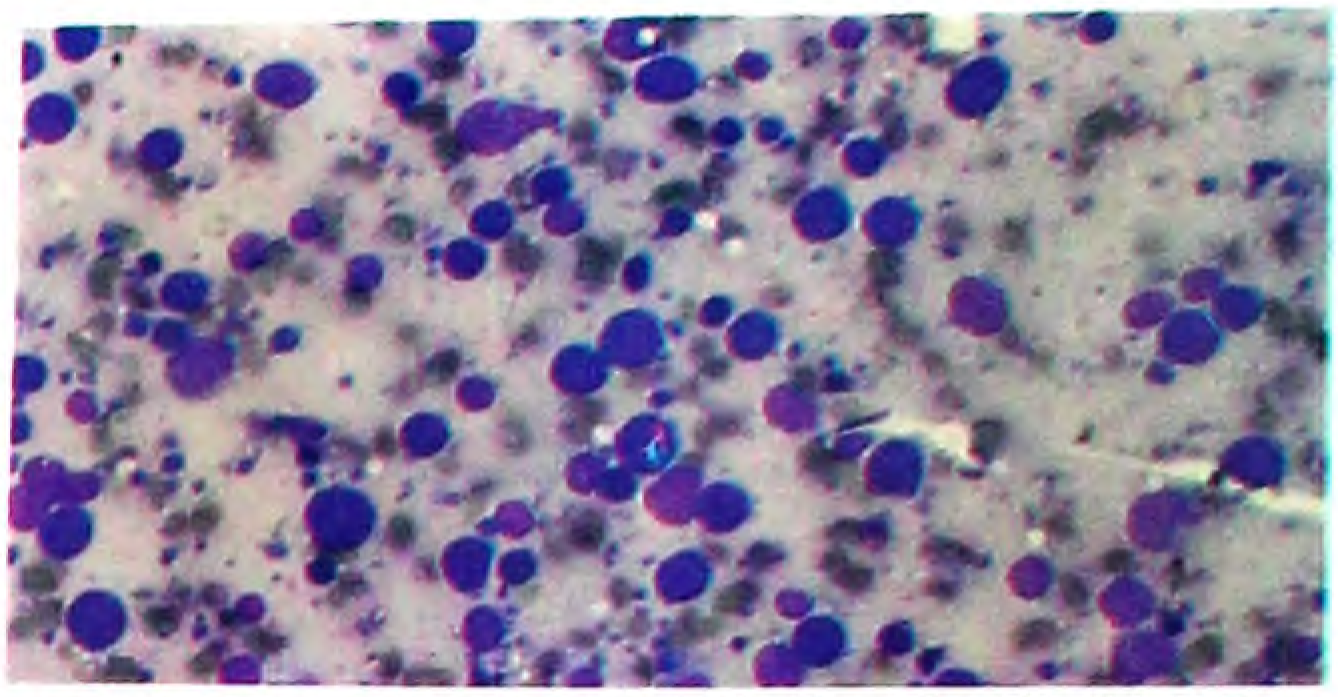

Fig.9: FNA - Lymphocytic thyroiditis (Case No. 50) with NHL large cell) - smears show pleomorphic lymphoid infiltrate similar to reactive lymphnode MGG x 400.

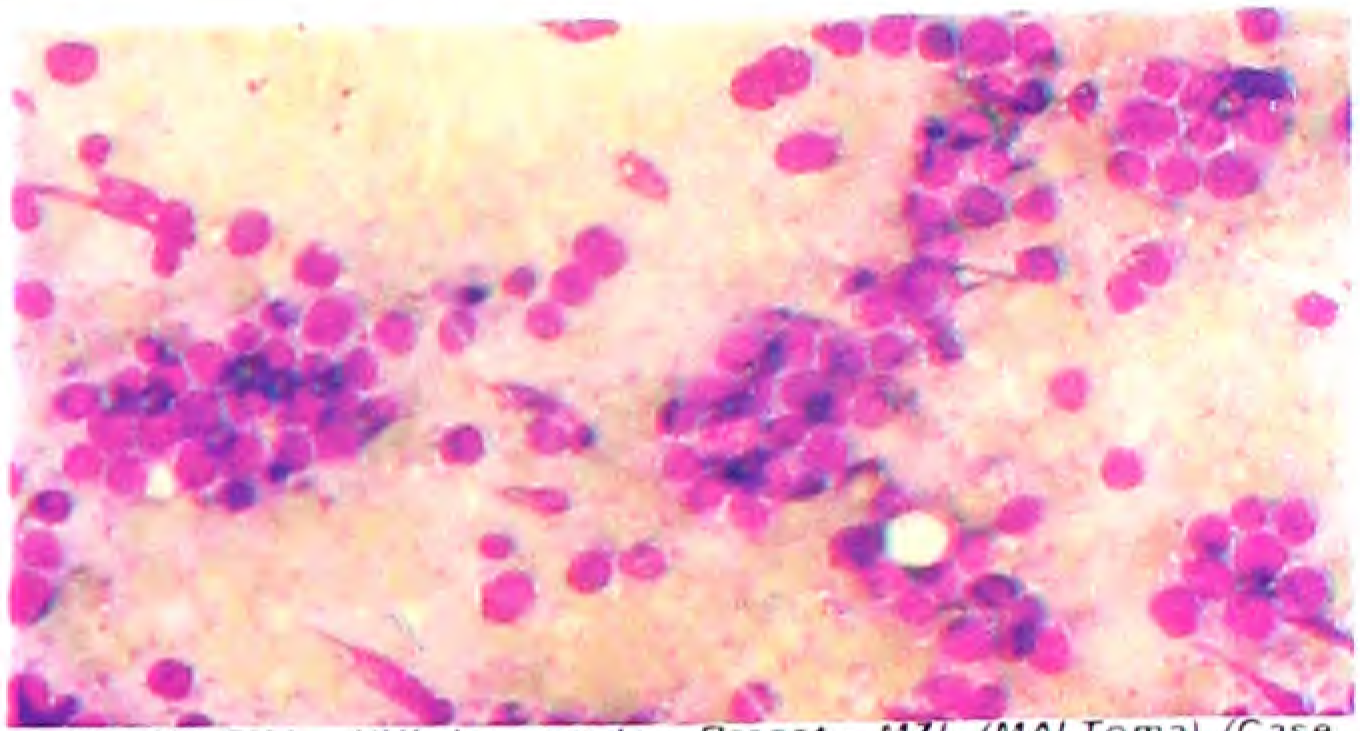

Fig.10: FNA - NHL low grade - Breast - MZL (MALToma) (Case 46) - shows duct epithelium infiltrated by small lymphoid cells MGG x 400.

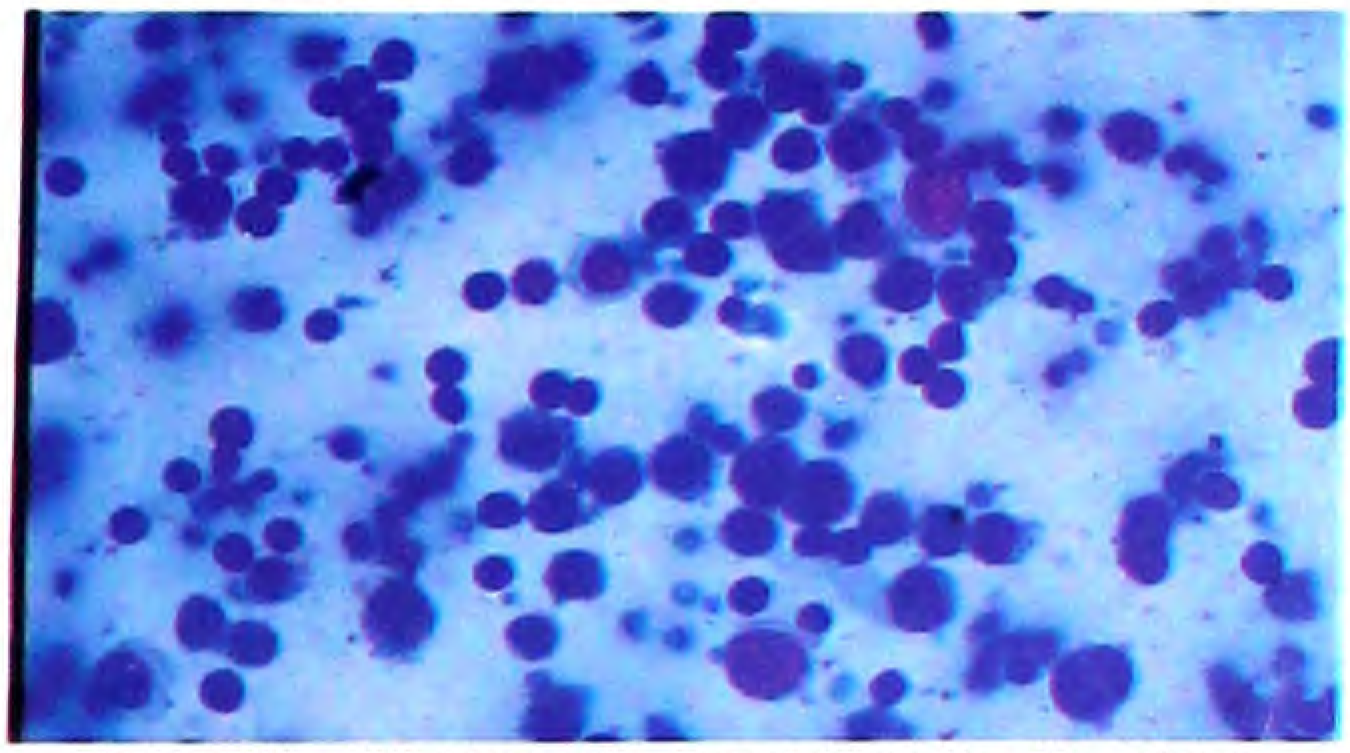

Fig.11: FNA - NHL large cell - Breast (Case No. 23) - shows large lymphoid cells dispersed and small loose clusters of 3-4 cells. MGG x 400.

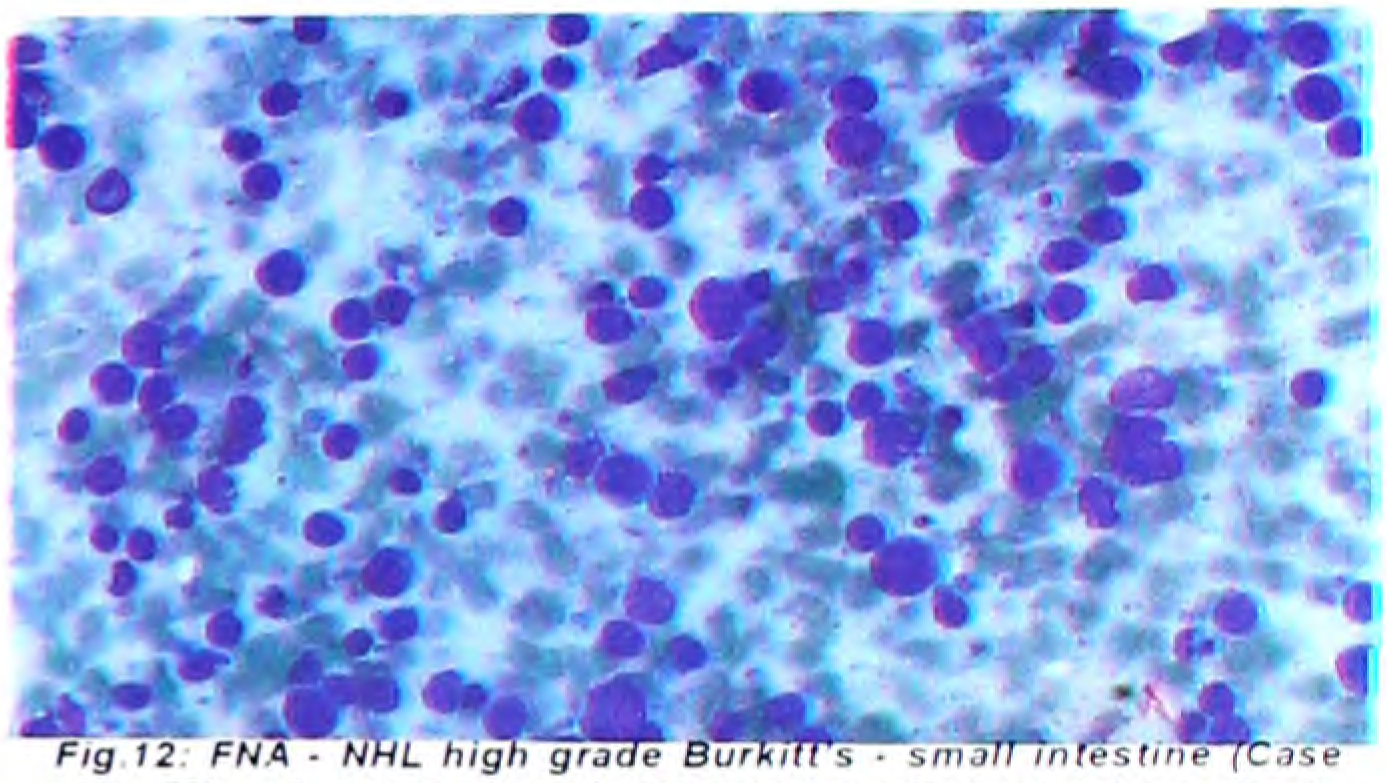

Fig.12: FNA - NHL high grade Burkitt's - small intestine (Case No. 58) - shows sheets of dispersed medium sized lymphoid cells and interspersed tingible body macrophages MGGx400

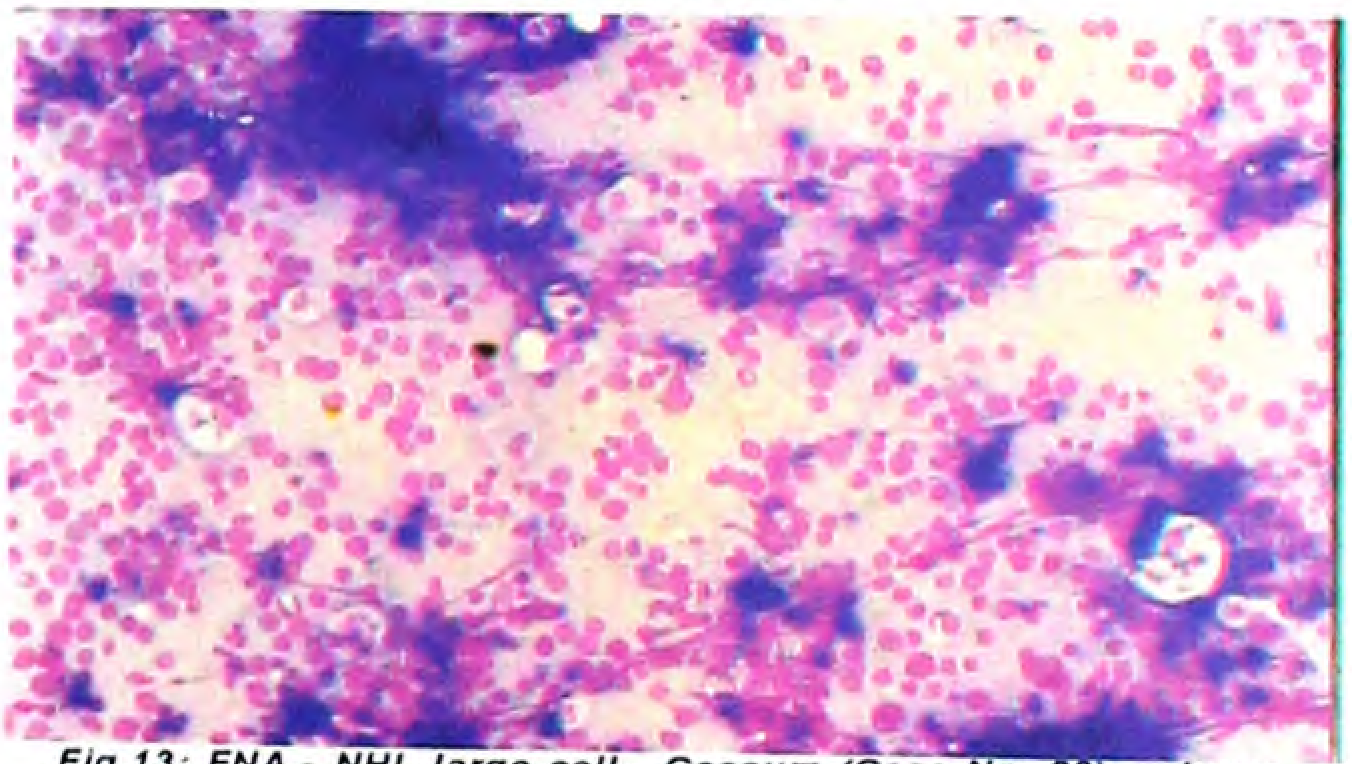

Fig.13: FNA - NHL large cell - Caecum (Case No. 63) - shows large non-cleared cells with some small lymphoid cells - MGGx400.

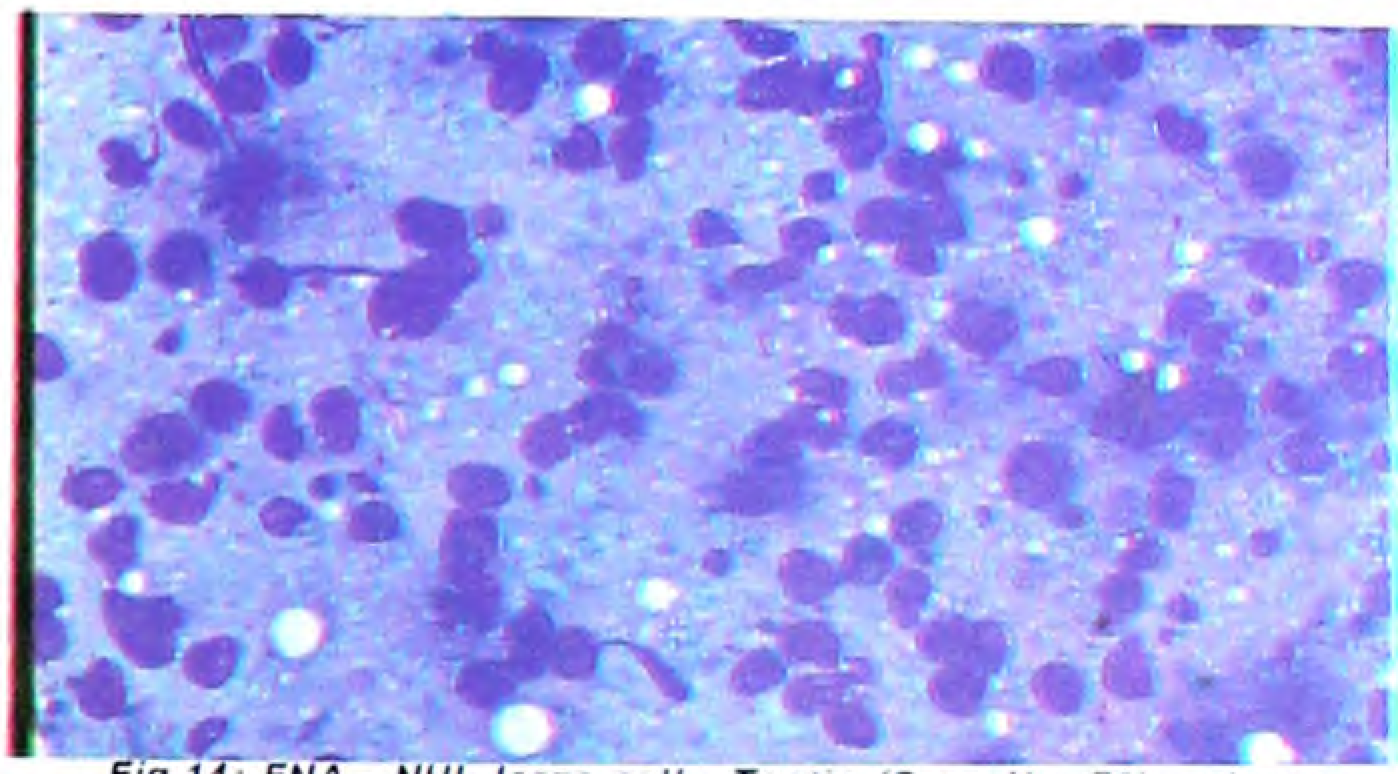

Fig.14: FNA - NHL large cell - Testis (Case No. 70) - shows large lymphoid cells despersed with some small lymphoid cells. MGG x 400.

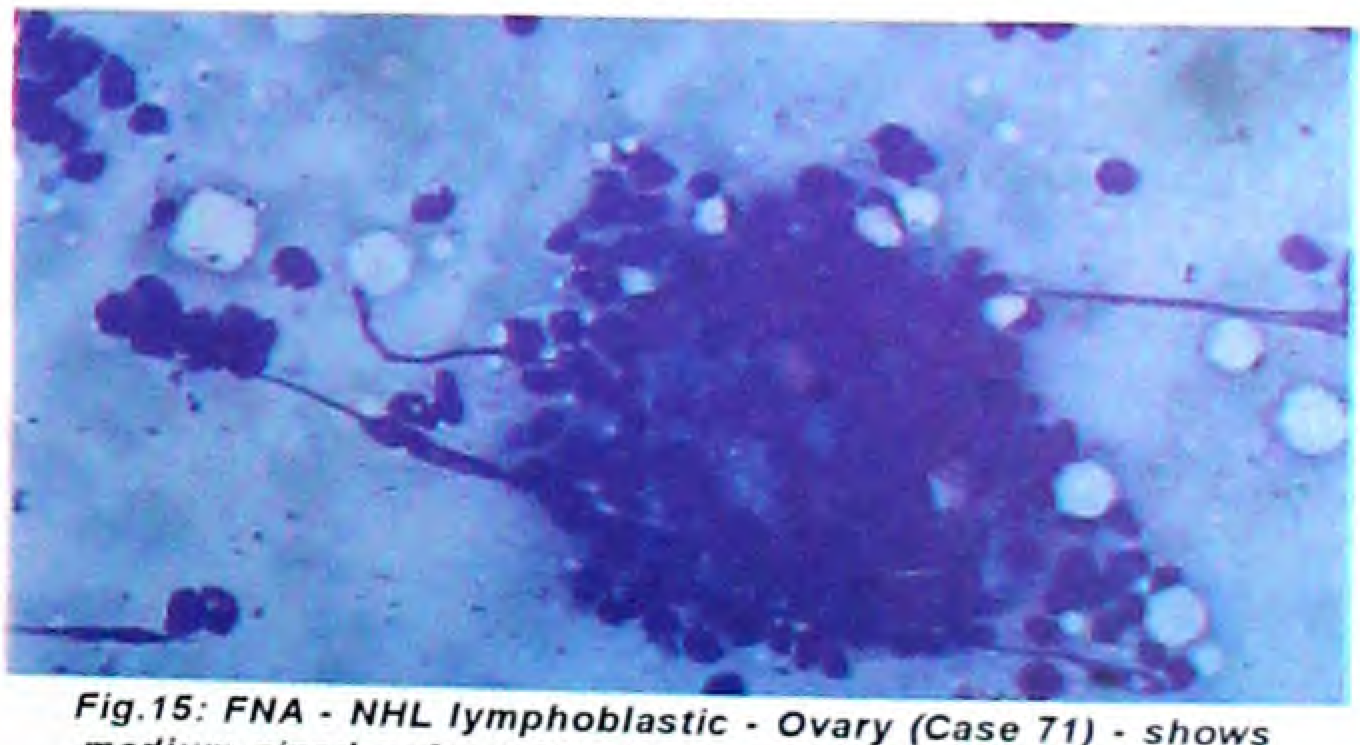

Fig.15: FNA - NHL lymphoblastic - Ovary (Case 71) - shows medium sized unform lymphoblasts, discrete and in small groups. MGG x 400.

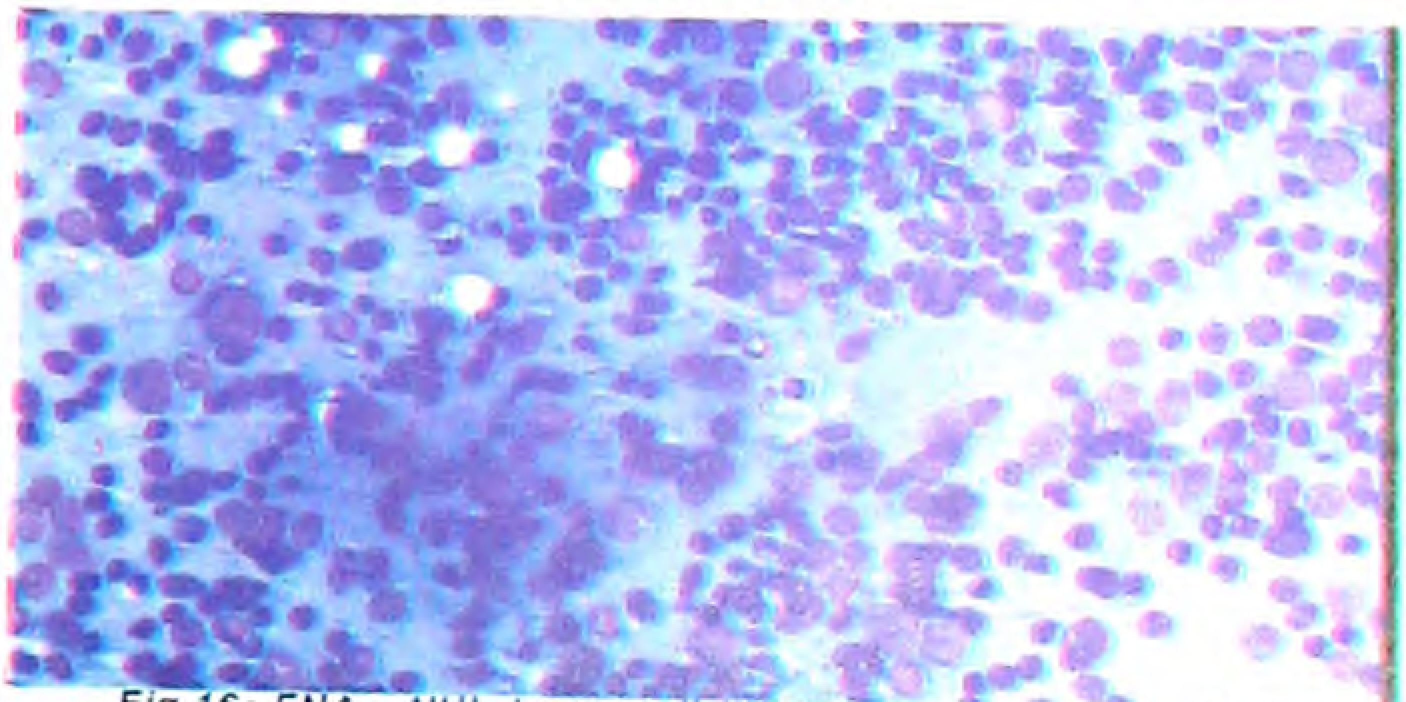

Fig.16: FNA - NHL large cell-illiac bone (Case 77) - shows dispersed large lymphoid cells with many lymphoglandular bodies MGG x 400.

July'2002), to make a total of 77 cases for the analysis.

The material and methods have been discussed in Part-I & II of this series. FNAC was conducted directly from the palpable lumps whereas non-palpable lesions were aspirated under imaging techniques like ultrasound and CT scan. Most of the patients were aspirated by one of the authors from the private clinic (ARK) and a small percentage were reported by the same author in the Pathology Department of Government Medical College, Srinagar.

The cytologic criteria applied for the diagnosis of lymphoma have been discussed in the part-I of this series of articles.(22) The histopathology was available in most of the cases Working Formulation(23) modified was applied both in FNAC and histopathology to accommodate some entities like Maltomas Mantle cell lymphomas and lymphocyte/histiocyte-rich B-cell lymphomas. However, the mixed cell category was lumped with large cell category.

RESULTS

A total of 77 extra nodal non-Hodgkin's lymphomas analyzed have been categorized into various groups viz. soft tissues, salivary glands, oral cavity, thyroid, breast, mediastinum and lung, gonads and bone (Table).

Soft tissue:

A total of 24 lymphomas were recorded from various sites like temporal region, scalp, cheek, tarsal plate, periorbit, maxilla, chin, chest wall and thigh. Four cases were seen below the age of 14 and the remaining 20 cases showed wide age-range between 20 and 80 years. There were 18 males and 6 females. The cytologic diagnosis was small lymphocytic lymphoma (3 cases, Fig. 1), plasmacytoma (1 case, Fig. 2), large-cell lymphoma (14 cases, Fig. 3), LBL (4 cases) and sarcoma - NOS (2 cases). The histopathology was available in all but one case (case 24), who could not be followed subsequently. The histopathology confirmed cytologic findings. However, 2 cases (case 19 and 22, table) reported as sarcoma NOS on FNAC proved to be sclerosing large cell lymphoma and small non-cleaved cell lymphoma respectively. The plasmacytoma cheek (case 7) was a solitary lesion, without any evidence of multiple myeloma, para-proteinemia or underlying bone involvement.

Salivary Glands:

There were 4 cases, 3 from the parotid and 1 from submandibular salivary gland. From the parotid two male patients of 10 and 16 years were diagnosed as LBL. One of these later proved to have leukemia (LBL). Two other cases diagnosed as large cell lymphoma on FNAC proved to be large cell lymphoma and marginal zone (MALT) lymphoma in transformation to large cell lymphoma on histopathology (Fig. 4).

Oral Cavity:

There were 3 cases in this group, one from the palate in a 35 year old female and 2 from tonsils in 25 and 60 year old males. The cytologic diagnosis of large cell lymphoma (Fig. 5,6) in all these cases was confirmed by histopathology.

Thyroid Gland:

A total of 14 cases were recorded in this group with a wide age range of 19-80 years. There were 10 females and 4 males. Eight cases had involvement of both the lobes, four cases had right lobe involvement and 1 case each involved left lobe and isthmus. The cytological diagnosis was lymphoplasmacytoma (1 case), suggestive of MZL (1 case, Fig. 7) and large cell lymphoma (12 cases). One case (Case 50) showed large cell lymphoma and lymphocytic thyroiditis from two different sites on FNA (Figs. 8 & 9). Surgical histopathology was available only in one case (Case 41) which confirmed the diagnosis of large cell lymphoma. Subsequent investigation showed these as Stage I/II lymphomas at Sher-i-Kashmir Institute of Medical Sciences, Soura, Srinagar (SKIMS).

Breast:

There were seven cases in this group with a wide age range of 25-60 years with most cases in third and fourth decade. There were 6 females and 1 male. The cytologic diagnosis was small cell lymphoma (1 case, Fig. 10), large cell lymphoma (5 cases, Fig. 11) and sarcoma NOS (1 case). The histopathology was available in all but one case (Case 52) which could not be traced. The histopathology showed marginal zone lymphoma (1 case), follicular and diffuse lymphoma (1 case) and large cell lymphoma (4 case, Figs. 10-11). In the large cell category there was one case of large cell lymphoma with lot of sclerosis, yielding scanty material on FNA, explaining mis-diagnosis. One case (Case 46) was a stage-I disease, who after 10 year follow-up is doing well. The remaining 5 cases had stage-II and III disease at the time of presentation. Case 48 was in last trimester of pregnancy. She subsequently died of uncontrolled disseminated lymphoma two years after diagnosis. Case 50, a 45 year female, was a known case of NHL-colon treated one year earlier in 1999. She presented with NHL breast in the year 2000 and three months later with NHL left-thigh.

Mediastinum and lung:

There were two cases in this group, one each from mediastinum and lung, both aspirated under CT-guidance by the Radiologist. Anterior mediastinum lesion was a typical T-cell lymphoblastic lymphoma with pleural effusion in a 19-year male. The trucut biopsy confirmed this as lymphoblastic lymphoma. The second case was a 45 year male with cytologic diagnosis of large cell lymphoma. No open biopsy was available in this case. He was treated at PGI Chandigrah in the year 2001 with a good response but relapsed with lymphoma abdominal nodes in the year 2002.

Gastrointestinal Tract:

There were 14 cases in this group 12 from large intestine and 2 from small intestine. No case was aspirated from stomach normally the commonest site. Of the gastrointestinal lymphomas there were 2 cases below 14 years in males with the cytologic diagnosis of high-grade lymphoma (Burkitt's lymphoma) (Fig. 12). The remaining 12 cases showed a wide age range of 16 to 80 years including 10 males and 2 females with two cases from caecum and ten cases from colon. The cytologic diagnosis was large cell lymphoma in all the 12 cases (Fig. 13). Histopathology was available in 13 cases (Table). All the cases with the exception of three cases (Case 5, 64 and 66) were localized lesions at the time of presentation.

Gonads:

There were 2 cases from testis in 45 and 70 year old males both with unilateral involvement. The cytologic diagnosis was large cell lymphoma in one and seminoma in the other (table). On orchidectomy, histopathology of both the cases proved to be large cell lymphomas (Fig. 14).

There was one case of a 13 year old female with ovarian mass aspirated under USG, diagnosed as lymphoblastic lymphoma (Fig. 15). The laprotomy revealed disseminated lymphoma abdomen (Figs. 15 & 13).

Bone:

There were 6 cases of NHL bone with a wide age range of 30-70 years including 4 males and 2 females. The cytologic diagnosis of lymphoma was offered in 5 cases (Fig. 16) and suspicion of lymphoma was raised in one case (Case 72). However, the histopathology available confirmed the diagnosis of large cell lymphoma in all. Only 2 cases (Case 72 and 73) were localized diseases at the time of presentation. All the lesions had local pain and lytic lesions. Two cases (case 74 and 76) had soft tissue involvement as well.

DISCUSSION

Extra-nodal lymphomas vary in frequency from 26%[10] to over 40%[11] in different geographic locations. In the USA, the most frequent sites in decreasing order in histopathologic study were stomach, skin, oral cavity and pharynx, small-intestine, CNS, colon and rectum.(10) With the concept of MALT lymphomas and re-classification of pseudolymphomas and AIDS and iatrogenic immunosupression, these figures are expected to increase. The males generally predominate over females except in breast and thyroid.[10]

Seventy-seven extra-nodal lymphomas encountered in this study on FNAC, included from various sites like soft tissue (24 cases), salivary, glands(4), oral cavity(3), thyroid(14), testes(2), ovary(1) and bone(6). The cytologic criteria applied for the cyto-diagnosis were similar to those for nodal lymphomas.[22] The open biopsy was available for most of the cases to evaluate the FNAC results.

Working Formulation[23] for classification of lymphomas was applied on both FNAC and histopathology with slight modification to accommodate entities like marginal zone, mantle cell and lymphocye/histocytc-rich B cell lymphoma.

Publications on FNAC of extra-nodal lymphomas are comparatively few.(12-19) One of the recent report is that of Stewart et al(16) based on 25 extra-nodal lymphomas including skin and soft tissue (10 cases), thyroid (5 cases), salivary gland (5 cases), breast (4 cases) neck and pharynx (1 case each). They used ancillary studies like immunocyto-chemistry, ISH for light-chain mRNA and PCR for Ig heavy-chains to support and augment morphologic diagnosis. There was one false positive diagnosis in their study.

In the present study of 77 cases, 9 cases were seen below the age of 14 and those above 14 showed a wide age range between 16 and 90 years. However, majority of these (57/68) were seen in the age group 30 and above. There was an overall male predominance. However, in breast and thyroid there was female predominance and these together constituted more than half of the cases (16/29) in females.

On FNAC, the extra-nodal lymphomas were subtyped according to Working Formulation modified (V.S). Thus the analysis showed lymphoblastic lymphoma (8 cases), small lymphocytic lymphoma (3 cases), lymphoplasmacytoma (1 case), plasmacytoma (1 case), small cell lymphoma (1 case), marginal zone lymphoma (1 case), large cell lymphoma (55 cases), Burkitt's lymphoma (2 cases), sarcoma NOS (3 cases) and seminoma (1 cases). Histopathologic comparison was available in 59 cases including 6 cases of lymphoblastic lymphoma, 3 cases of small lymphocytic lymphoma, one case of plasmacytoma, 45 cases of large cell lymphoma, 3 cases of sarcoma NOS and the case of seminoma testis. Cytopathologic comparison was 100% accurate in 10 cases of lymphoblastic lymphoma, small lymphatic lymphoma and one case of plasmacytoma. Out of 45 cases of large cell lymphoma 43 proved to be large call lymphoma, 1 case follicular and diffuse large cell (Case 47) and 1 case

TABLE: FINE NEEDLE ASPIRATION CYTOLOGY (FNAC) OF EXTRA-NODAL LYMPHOMAS (77 cases)

SOFT TISSUE LYMPHOMAS (24)

i. Temporal Region

S.No.	Year	Age/Sex	Site	FNAC Dx.	Histopathology Dx.
1	1998	52/M	B/L	SLL	SLL
2	1999	8/M		LBL (High grade)	LBL
			ii. Scalp		
3	1994	10/F		LBL (High grade)	LBL
4	1996	40/F	Multiple nodules		LCL
5	2001	26/M	Multiple nodules	LCL	LCL
			iii. Cheek		
6	1995	60/F		LCL	LCL
7	2000	60/M		Plasmacytoma	Plasmacytoma
			iv. Tarsal plate		
8	1996	50/M	B/L	SLL	SLL
			v. Periorbital region		
9	1996	45/M		LCL	LCL
10	2002	70/M		SLL	SLL
			vi. Maxilla		
11	1996	5/M		LBL	LBL
12	1998	42/M	(+ palate)	LCL	LCL
13	2000	90/F		LCL	LCL
14	2000	3/M		LBL	LBL
15	2001	70/M		LCL	LCL
			vii. Chin		
16	2002	65/M		LCL	LCL
			viii. Chest wall		
17	1992	47/M	Posterior aspect	LCL	LCL
18	1992	45/M	Anterior aspect	LCL	LCL
19	1994	50/M	Anterior aspect	Sarcoma NOS	LCL
20	2000	20/M	Anterior aspect	LCL	LCL
			ix. Lower Extremity		
21	1995	58/F	Thigh	LCL	LCL
22	1996	30/F	Thigh	Sarcoma NOS	High grade small NCL
23	2001	50/M	Thigh	LCL	LCL
24	2001	88/M	Thigh	LCL	?
			x. Salivary glands (4)		
25	2000	10/M	(Parotid RC)	LBL	LBL - ALL
26	2000	60/M	(Parotid RC)	LCL	LCL
27	2001	16/M	(Parotid RC)	LBL	LBL
28	2002	35/F	(Sub-mandibular R)	LCL	MZL LBL
			xi. ORAL CAVITY (3)		
29	1997	35/F	(Hard palate)	LCL	LCL
30	2001	60/M	(Tonsil – Rt)	LCL	LCL
31	2002	25/M	(Tonsil – Lt)	LCL	LCL
			xii. THYROID (14)		
32	1995	40/F	Both lobes	LCL	-
33	1995	40/F	Rt. Lobe	LCL	-
34	1995	50/F	Both lobes	LCL	-
35	1996	35/F	Both lobes	Low grade ? MZL	-
36	1997	50/F	Isthmus	LCL	-
37	1997	50/F	Both lobes	LCL	-
38	1997	50/M	Lt. lobe	LPL	-
39	1998	58/M	Rt. Lobe	LCL	-
40	1999	60/M	Both lobe	LCL	-
41	2000	23/F	Rt. Lobe	LCL	LCL
42	2000	50/F	Both lobes	LCL+lymphocytic thyroiditis	-
43	2001	80/ F	Both lobes	LCL	-
44	2001	19/M	Both lobes	LCL	-
45	2002	19/F	Rt. Lobe	LCL	-

			xiii. BREAST (7)		
46	1991	30/F		Low grade small lymphoma	MZL
47	1994	33/F		Large cell lymphoma	Follicular & Diffuse LCL
48	1996	25/F (pregnant)		LCL	LCL
49	1996	30/F		LCL	LCL
50	1999	45/F		LCL	LCL
51	2000	60/F		Sarcoma (NOS)	LCL (Sclerosing)
52	2002	35/F		LCL	-
			xiv. MEDIASTINUM & LUNGS (2)		
53	2001	19/M	Anterior Mediastinum + Pleural effusion	LBL	LBL
54	2001	45/M	Lung	LCL	-
			GIT (14)		
55	1995	80/M	R. Colon	LCL	LCL
56	1995	65/M	R. Colon	LCL	LCL
57	1996	60/M	L. Colon	LCL	LCL
58	1998	5/M	Ileum	High grade NHL	-
59	2000	16/F	R. Colon	LCL	-
60	2000	40/F	T. Colon	LCL	LCL
61	2000	65/M	L. Colon	LCL	LCL
62	2001	80/M	L. Colon	LCL	
63	2001	40/M	Caecum	LCL	LCL
64	2001	12/M	Ileum	High grade (Burkitt's) lymphoma	
65	2001	48/M	R. Colon	LCL	LCL
66	2001	45/M	L. Colon	LCL	LCL
67	2001	70/M	L. Colon	LCL	LCL
68	2001	28/M	Caecum	LCL	LCL
			GONADS (3)		
69	1994	70/M	Lt. Testis	LCL	LCL
70	1994	45/M	Rt. Testis	Seminoma	LCL
71	2002	13/F	Ovary	LBL	LBL
			BONE (6)		
72	1991	30/F	Femur	? Large cell	LCL
73	1995	70/M	Radius (upper end)	LCL	LCL
74	1996	40/F	Rib & Soft tissue	LCL	LCL
75	2001	65/M	Ischium	LCL	LCL
76	2002	45/M	Rib & Soft tissue	LCL	LCL
77	2002	70/M	Iliac Bone	LCL	LCL

Key words : *SLL= Small lymphocytic lymphoma; LBL= Lymphoblastic lymphoma;*
LCL= large cell lymphoma; NOS = Not otherwise specified;
MZL = Marginal zone lymphoma

marginal zone (MALT) lymphoma with large cell transformation. Three cases of sarcoma NOS on histopathlogy proved to be large cell lymphoma with sclerosis from chest (Case 19) and breast (Case 51) and small non-cleaved cell lymphoma (Case 22). Case of seminoma testis (Case 70) also proved to be large cell lymphoma. Thus, there were 4 false negative cases in a total of 59 cases for which histopathology was available.

Lymphoblastic lymphoma smears were highly cellular composed of monotonous medium size blast cells with high nuclear cytoplasmic ratio, scanty basophilic cytoplasm, fine chromatin, indistinct nucleoli and occasional mitotic figures. Lymphoglandular bodies were present. Burkitt's lymphoma smears were also cellular with medium sized cells, more abundant basophilic cytoplasm with vacuolations and relatively more distinct nucleoli. There were many mitotic figures and tingible body macrophages (Fig. 12). Small lymphocytic lymphoma (Fig. 2) were highly cellular smears of monotonous small lymphoid cells with high nuclear-cytoplasmic ratio, clumpy chromatin and very scanty cytoplasm. Lymphoplasmacytic lymphoma smears showed a mixture of dispersed cells composed of lymphocytes, lymphoplasmacytoid and plasma cells. Plasmacytoma (Fig. 1) showed mixture of mature and immature plasma cells. Large cell lymphoma showed fairly cellular smears with large lymphoid cells as the main cell component (Figs. 1,4,6,8,11 & 16). However, sclerosing large cell lymphoma was a problem due to scanty material comprising of dispersed large cells with no clue to lymphoid origin.

In summary, therefore, diagnosis of extra-nodal lymphomas in FNAC smears depends on the type of lymphoma. High grade blast and large cell lymphomas are easy to interpret most of the times. Poor yield due to sclerosis may account for mis-diagnosis. However, immunocyto-chemistry can help in these cases. Small lymphocytic lymphomas and lymphoplasma cytomas are not difficult to interpret but need caution to rule out reactive lymphoid population. Confirmation of the monoclonality can secure the diagnosis. Plasmacytoma - extra-skeletal is made up of mature and immature plasma cells without any inter-mixture of neutrophils, eosinophils and histocytes. Marginal zone or MALT lymphoma are not easy to interpret with confidence on morphology alone. Even suspicion of lymphoma in these cases can be supplemented by ancillary studies or excision biopsy.

REFERENCES

01. Greig EDW, Gray ACH. Note on the lymphatic glands in sleeping sickness. Brit Med J 1904; 1: 1252.

02. Zajicek J. Aspiration biopsy : Cytology of supra-diaphragmatic organs. New York S Karger, 1974.

03. Zajicek J. Aspiration biopsy : Cytology of infradiaphragmatic organs. New York S. Kargar, 1979.

04. Carter T, Feldman P, Innes D, et al. The role of cytology in the diagnosis of lymphoma. Acata Cytol 1988; 32: 846-53.

05. Das DK, Gupta SK, Dutta BN, Sharma SC. FNA diagnosis of non-Hodgkin's lymphoma and its subtyping under Working Formulation in 175 cases. Diag Cytopathol 1991; 7: 487-98.

06. Young NA, Al Saleem TI, Al Saleem Z, et al. The value of transformed lymphocytic sub-classification of non-Hodgkin's lymphoma by fine needle aspiration. Am J Clin Pathol 1997; 108: 143-51.

07. Hughes JH. Katz RL, Fouseca GA, Cabanilla FF. Fine needle aspiration cytology of mediastinal non-Hodgkins, non-lymphoblastic lymphoma. Cancer 1998, 84(1): 26-35.

08. Jeffers MD, Milton J, Herriot R, Mckean N. Fine needle aspiration cytology in the investigation of non-Hodgkin's lymphoma. J Clin Pathol 1998;51(3): 189-96.

09. Wakely PE (Jr): Fine needle aspiration cytopathology of malignant lymphoma. Clin Lab Med 1998; 18: 541-59.

10. Greiner TC, Medeiros LJ, Jaffe ES. Non-Hodgkin's lymphoma. Cancer supplement 1995; 75(1): 370-80.

11. Crawford JM. The gastrointestinal tract. In: Robbin's Pathologic Basis of Disease. 6th edition, W.B Saunders Company pp 837-38.

12. Tane E, Shoog L. Fine needle aspiration cytology and immunocyto chemistry in the diagnosis of lymphoid lesions in the thyroid gland. Acta Cytol 1989; 33(1): 48-32.

13. Sterret G, Whitaker D, Shilkin KB, Walters N. The fine needle aspiration cytology of mediastinal lesions. Cancer 1983; 51: 127-35.

14 Carter TR, Feldman, Jnnes Jr, Frierson Jr HF, Frisy AL. The role of Fine Needle Aspiration Cytology in the Diagnosis of Lymphoma. Acta Cytol 1987; 22(6): 848-53.

15 Dunphy CH, Ramos R. Combining Fine Needle Aspiration and Flow Cytometric Immunophenotyping in evaluation of nodal and extra-nodal sites for possible lymphoma : A retrospective review. Diagn Cytopathol 1996; 16(3): 200-06.

16. Stewart CJ, Jackson R, Farquhorson M, Richmohd J. FNAC of extranodal lymphoma. Diag Cytopathol 1998; 19(4): 200-06.

17. Dey P, Luthra UK, Sheikh ZA, Mathews SB. Fine Needle Aspiration Cytology of primary non-Hodgkin's lymphoma of the tongue - A case report. Acta Cytol 1999; 43(30): 422-24.

18. Park IA, Kim CW. FNAC of malignant lymphoma in an area with high incidence of T-cell lymphoma. Correlation of accuracy of cytologic diagnosis with histologic sub-type and immunophenotype. Acta Cytol 1999; 43(6): 1059-69.

19. Matsushima AY, Hamele-Bena D, Osborne BN. Fine needle aspiration biopsy findings in marginal zone B-cell lymphoma. Diag Cytopathol 1999; 20(4): 190-98.

20. Meda BA, Buss DH, Woodruff RD, Cappellari JO, Rainer RO, Powell BL, Geisinger KR. Diagnosis and sub-classification of primary and recurrent lymphoma. The usefullness and limitations of fine needle aspiration cytopathology and flow cytometry. Am J Clin Pathol 2000; 113(5): 688-99.

21. Cannon CR, Richardson D. Value of flowcytometry with fine needle aspiration biopsy in patients with head and neck lymphomas. Otolaryngeal, Head and Neck Surgery 2000; 123(6): 696-99.

22. Khan AR, Omar Shuab, Rashid Sumia, Besina Syed. Utility of fine needle aspiration in the diagnosis of nodal primary non-Hodgkin's lymphoma (Part-I). JK Pract 2002; 9(3): 197-204.

23. The Non-Hodgkin's Lymphoma Pathologic Classification Project Committee. National Cancer Institute Sponsored Study of Classification on Non-Hodgkin's Lymphoma. Summary and Description of Working Formulation for Clinical Usage. Cancer 1982; 49: 2112-35.

24. Carlson R, Gleeson M, Eveson J. Mesenchymal lymphoreticular metastatic and periglandular tumors and the uncommon types of salivary gland tumors. In: The Pathology and Surgery of the Salivary Glands. ISIS Medical Media, Oxford 1997; pp 17-90.

25. Freeeman C, Berg JN, Cutler SJ. Occurrence and prognosis of extra-nodal lymphoma. Cancer 1972; 29: 252-60.

26. Singer JA. Primary lymphoma of the thyroid. Ann Surg 1998; 64: 334-37.

27. Hamburger JI, Malter JM, Kine SR. Lymphomas of the thyroid. Ann Inter Med 1983; 99: 685-93.

28. Isaacson PG, Norton AJ. Extranodal lymphomas Churchill Livingstone Edinburgh, pp 103-16, 1994.

29. Babrow LG, Richards MA, Happerfield LC. Breast lymphoma - A Clinicopathologic Review. Human Pathol 1993; 24: 274-278.

30. Arber DA, Simpson JF, Weiss LM, Rappapert H. Non-Hodgkin's lymphoma involving breast. Am J Surg Pathol 1994; 18: 288-93.

31. Crawford JM. The gastrointestinal tract. In: Robbin's Pathologic Basis of Disease. WB Saunder's Company 1999; pp 837-38.

32. Anderson JR, Armitage JO, Weisenberger DD. Epidemiology of the non- Hodgkins lymphoma. The distribution of the major sub-types differ by geographic location. Non-Hodgkin's lymphoma. Classification Project. Ann Oncology 1998; 9: 717.

33. Paladugh RR, Bearman RM, Rappaport H. Malignant lymphomas with primary manifestation in the gonad. A histopathologic study of 38 cases. Cancer 1980; 45: 551-57.

34. Khan AR, Chowdhary ND. Testicular lymphoma - A histopathologic study of 12 cases. JK Pract 1999; 6(3): pp 220-22.

35. Dalla-Faverg R, Gaidano G. Lymphoma. In: Cancer Principles & Practice of Oncology. Eds. Deista (Jr). VT. Helman S. Rosenberg SA. Lipincott. Williams & Wilkins 2000; pp 2300-2303.

36. Unni KK, Inwards CY. Tumors of the osteoarticular system. In: Diagnostic Histopathology of tumours. Ed. Fletcher CDM. Churchill Livingstone 2000; pp 1547-48.

BRONCHIAL ASTHMA

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Definition

Bronchial Asthma is a chronic inflammatory disease of airways characterised by recurrent wheezing; Cough (Worse at night), recurrent shortness of breath and chest tightness. Diagnosis is suggested by reversible airways obstruction on Spirometry performed before and after the administration of a short acting bronchodilator. Airway obstruction is indicated by a decreased forced expiratory volume in one second (FEV_1) and a decreased ratio of FEV_1 to forced vital capacty (FVC) relative to predicted values while reversibility is indicated by an increase in FEV_1 by 12-15% after short acting bronchodlator therapy in form of inhalation. In patients with asthma symptoms and normal Spirometry, an assessment of the diurnal variation in peak expiratory flow (PEF) of more than 20 percent helps in establshing the diagnosis.

Prevalence

World wide 100 to 150 million people, roughly equivalent of the population of Russian federatiion, suffer from Bronchial Asthma and the deaths have reached 1,80,000 annually. India has an estimated 15 to 20 million asthmatics. In India, rough estimates indicate a prevalence of between 10 to 15 percent in 5-11 years old children.

Pathophysiology

Hallmark of Bronhial asthma is airway inflammatory. Genctics and an environmental insult both play a role. It has been seen that an incidence of asthma is six times more in the newborn whose both patents are asthmatic as compared to the normal parents. Envronment factor like weather changes, exposure to allergens as pollen, dust, animal dander, smoke, cold, exercise and isocyanates etc. initate the inflaimmaitory process. At the cellular level events begin when macrophages present antigen to T-iymphocytes that elaborate type 2T-helper (TH^2) cytokines such as interleukins[4,5] and 13. L-4 and 13 regulate the IgE synthesis which crosslink with mast cells and cause their disintegration resulting in release of mediators like histamine, leukotrienes, prostaglandins and inflammatory cytokines like TNF-a. In addition, these interleukines increase the adhesion molecular receptors on vessel walls as well. IL-5 mediates eosinophil recruitment and B-chemokines which are considered pivotal cellular mediatiors of asthma. Increased vascular cell adhesion molecules and intercellular adhesion molecules-1 and 2 along with themediators released from eosinophils result in remodelling which s characterised by epithelial cell denudation, deposition of such matrix proteins as laminin, tenascin, fibrin, fibronectin and type IV and V collagen and hyperplasia of smooth muscles and goblet cells. At the intracellular level, NF-Kappa B and ieukotrenes play a pivotal role in airway inflammatory and are therapeutic points of attack. NF-Kappa B is a DNA binding protein that functions as a transcription factor and controls the 'readout' of genes while leukotrienes are the products of arachidonic acd metabolism and include LT-A4 to LT-E4. LT-D4 is the most potent and LT-E4 has the longest activity.

Classification

An expert panel for the National Asthma Education and Prevention Programme (NAEPP) recently issued new guide-lines for asthma. Based on these guidelines asthma is classified as:

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CLASSIFICATION	SYMPTOMS	NIGHT SYMPTOMS	LUNG FUNCTION
STEP 1 Mild intermittent Asthma.	•Symptoms occur thrice a week or less. •No symptoms and PEF normal in between. •Brief exacerbation.	•Symptoms occur no more than twice a month.	$\frac{FEV1}{FVC} > 80\%$ PEF variability $< 20\%$
STEP 2 Mild Persistent Asthma	•Symptoms occur twice a week or less. •Exacerbation may affect activity	•Symptoms occur more than twice a month.	$\frac{FEV1}{FVC} > 80\%$ PEF variability $< 20\text{-}30\%$
STEP 3 Moderate Persistent Asthma	•Daily symptoms. •Daily use of inhaled B2 agonists. •Exacerbation affect activity	•Symptoms occur more than once a week	$\frac{FEV1}{FVC} > 60\text{-}80\%$ PEF variability $> 30\%$
STEP 4 Severe Persistent Asthma	•Continual symptoms •Limited physical activity •Frequent exacerbation	•Frequent symptoms	$\frac{FEV1}{FVC} > 60\%$ PEF variability $> 30\%$

Management

Asthma is not a curable disease and the best management lies in its prevention. The patients should be educated properly regarding the course of disease; prevention of various preciptating factors in the form of environmental control and also the management of complicating factors like sinusitis, gastro esophageal reflux etc. As for the drug therapy is concerned, current options of medical management can be divided into

I) Quick relief medications / rescue medication.
II) Long term control medications.

I) *Rescue Medication* are used in acute state and have an early onset and short duration of action. These include:

a) Short acting Bronchodilators:

B2 agonists	-	Albuterol
	-	Pirbuterol
Anticholinergics	-	Ipratropium
Combination	-	Albuterol and Ipratropium

b) Systemic steroids

II) Long term medications are used in maintenance phase and have longer duration of action. These include:

a) Long acting Bronchodilators

B2 agonists - Salmeterol

b) Anti Inflammatory Drugs

Non Steroidal	-	Cromolyn sodium
	-	Nedocromil
Steroidal	-	Beclamethasone, Triamcinolone, Budesonide, Flunside, Fluticasone and Fluticasone Plus Salmeterol.

Non Steroidal anti inflammatory drugs appear to be more effective in inhibiting bronchospasm induced by exercise, cold air and provocative testing. They are safe in children and pregnant woman.

c) Leukotriene modifiers

Leukotriene blockers	-	Montelukast
	-	Zafirlukast.
5-LPO inhbitors	-	Zileuton.

Zafirlukast is appropriate for children more than seven yeas of age while Montelukast is appropriate for children more than two yeas of age. These drugs must be taken in empty stomach and watch for interaction with other drugs is required.

d) Phosphodiesterase inhibtors

Theophylline, Aminophylline and other Theopylline salts. In addition to the inhibition of phosphodiesterase, blockade of adenosine receptor as well as anti inflammatory role has also been documented.

NAEPP emphasises upon an aggressive 'step care' approach therapy i.e. therapy is instituted at a step higher than the patients current level of asthma severity with the gradual 'step down' once control is achieved.

Step Care Approach

Step-1

Mild intermittent asthma - No daily medication

Step-2

Mild persistent asthma	-	One daily medication (anti inflammatory drug)
	-	Low dose inhaled steroid

	/nedocromil - Children usually begin with the trial of nedocromil/cromolyn. - zafirlukast/ zileuton >12 years. *(OR)* Sustained release theophylline and plasma drug level should be between 5-15 ug/ml.
Step-3 Moderate persistent asthma	- One daily medication - Medium dose inhaled steroids (OR) Two daily medication Low-medium dose inhaled steroids + Long acting Bronchodilator.
Step-4 Severe Persistent Asthma	- Daily medication High dose inhaled steroid + Long acting Bronchodilator + Oral steroid in dose of 2 mg/kg/day with daily dose not exceeding 60mg.

Novel Therapies

Various agents being tested in early phase clinical trials are:

1) **Third generation B2 -agonists:**
An endogenous adrenaline is an R-isomer and all the synthetic selective B2-agonists are the mixture of R-isomer and S-isomer. R-isomer is a bronchodilator while S-isomer paradoxically cause bronchoconstriction. Therefore, a third generation B2-agonists in form of a pure R-isomer of Albuterol has been synthesised which causes bronchodilation only without any paradoxical bronchoconstriction.

2) **IgE Antagonists:**
Rhu Mab-E25 is a humanised chimeric monoclonal antibody directed to high affinity receptor-binding domain of human IgE. With its use there occurs diminished early and late phase response to allergens, diminished sputum eosinophilia, decreased airway hyper responsiveness and decreased levels of relevant receptor (Fc-Epsilon R) in mast cells.

3) **Cytokine Antagonists:**
Co-administration of anti IL-4 and anti IL-5 inhibits an IgE production, decrease airway eosinophilia and also hyperresponsiveness. Administration of soluble IL-4 receptor (SIL-4R) removes available IL-4 and has been tried in phase 2 clinical trials in humans.

4) **Adhesion molecule antagonist:**
Treatment with monoclonal antibody to ICAM-I decrease the infiltration and markedly attenuates the increase in airway responsiveness. Blockade of VCAM-I likewise leads to impaired movement of inflammatory cells.

5) **Transcription factor antagonists:**
NF-Kappa B is an intracellular molecule critical for expression of both cytokine and adhesion moldcules that functions as a transcription factor controlling the 'read out' of genes. Potential blockers of this critical molecule include:
   - Cyclosporin A
   - FK- 506
   - Aspirin- Triptolide
   - Serine protease inhibitors and
   - 4-trifluoro methy l derivative of salicylate.

6) **Oligonucleotide and Gene therapies:**
Vaccination with immuno-stimulatory DNA - sequence may be a way of modulating skewed TH2 response seen in atopic asthma. Since asthma is a polygenic disease, an 'epigenetic therapy' rather than gene replacement seems to be more practical. In epigenetic therapy, various non-sense codons are incited in the m-RNA so that the protein synthesis is abnormal.

7) **Gravitational surgery of blood**
Plasmapharesis and hemosorption using absorbents like KAU, SKN gemofer etc. are the two procedures Plasmapharesis permits to achieve more rapid but less durable remission and should be done on three procedure basis only. Effects start at first post-operative week and peaks at 7-14 days.

8) **Intermittent increasing normobaric isocapnic hypoxia:**
Hypoxic training (Yoga) positively influences the specific and non-specific immunological status and appears to

be associated with a far better stimulation of lymphocytes and neutrophils.

9) Neuropharmacological treatment:

Antidepressant TIANEPTINE has been tried in bronchial asthma. It has been seen that levels of free serotonin in plasma are increased in symptomatic patients with asthma. Tianeptine is the only drug known to reduce the level of free serotonin in plasma and also enhance the uptake by platelets.

10) Transdermal formulation

Transdermal formulation of Tulobuterol using the so called 'crystal Reservoir system' has been tried. This is the first transdermal chrono-delivery system reported anywhere in the world. It is effective and safe in children as well.

11) Radiotherapy

It has been in use since 1930. But because of increased incidence of breast cancer, this mode of therapy did not gain appreciation. However, with the usage of low dosage technique in which only 250 R is applied, the incidence of breast cancer has decreased markedly. Half of the radiation is applied to one large anterior and half to one large posterior mediastinal field.

12) Aerophytotherapy with olesan oil:

It has been tried in chronic obstructive bronchitis and bronchial asthma.

13) Saiboku-To:

It is a herbal medicine and have an antiasthmatic effect through suppression of type IV allergic reaction. Flavonoids and lignans have been implicated in its antiasthmatic effect.

14) Su-JOK therapy

It is one of the methods of alternative medicine and has been found to be efficacious in bronchial asthma.

References:

1. Bender SB, Vizel AA. Current Approach to the treatment of acute bronchial ashtma. Ter Arkh: 2000;72(8):10-12.
2. Borish LC et al. Interleukin-4 receptor in moderate a topic asthma. A phase I/II randomised, placebo-controlled trial. Am J Respir Crit Care Med 1999;160:1816.
3. Bryan SA et al. Effects of recombinant human interleukin-12 on eosinophils, airway hyper-responsiveness, and the late asthmatic response. Lancet 2000;356:2149.
4. Busse WW, McGill KA, Horwitz RJ. Leukotriene pathway inhibtors in asthma and chronic obstructive pulmonary disease. Clin Exp Allergy 1999;29(Suppl 2):110.
5. Ellingsen I, Florraag E, Andereassen AH et al. Plasma Pharesis in the treatment of steroid-dependent bronchial asthma. Allergy 2001;56(12):1202-5.
6. Kato H, Nagata O, Yamazaki M et al. Development of transdermal formualtion of tulo butterol for treatment of Bronchial Asthma. Yakugaku Zasshi 2002-112(1):57-69.
7. Krishna Swami G et al. Molecular therapies for asthma. Fed Practitioner 1999;2:16.
8. Krishna Swami G Treatment strategies for Bronchial asthma: and update. HOSP Pract (Off Ed)15: 2001;36(8):25-6, 29-35.
9. Leckie MJ et al. Effects of an interleukin-5 blocking monoclonal antibody on eosinophils, airway hyper-responsiveness, and the late asthmatic response. Lancet 2000;356:2144.
10. Lechin F. Vander Dijs B, Orozco B et al. Neuropharmacologic treatment of bronchial asthma with the antidepressant tianeptine: a double blind, corssover placebo-controlled study. Clin Pharmacol Ther 1998;64(2):223-32.
11. Marinin VF, Ishina TI, Fomina IG. Aerophyto therapy with olesan oil in the treatment of patients with bronchial asthma Klin med (MOSK): 2001;79(4):54-6.
12. Metzger WJ, Nyce JW. oligonucleotide therapy of allergic asthma J Alergy Clin Immunol 1999;104(2Pt-1):260.
13. Milgrom H et al. Treatment of allergic asthma with monoclonal anti IgE antibody. Rhu MAb-E25 Study Group. N Engl J Med 1999;341:1996.
14. Wong WS, Koh DS. Advances in immunopahrmacology of asthma. Biochem Pharmacol 2000;59:1323.

HIV-1 VACCINE NOT YET IN SIGHT

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A paper reviewing the status of HIV-1 vaccines was published in JK-Practitioner n 1997[1]. At that time there were high hopes that a vaccine for AIDS would be developed in the near future although the paper mentioned a mathematical study by Nowak et al that was not so optimistic.[2] Today five years later a vaccine for AIDS is still nowhere in sight. David D Ho and Yaoxing Huang of Rockfeller University have in a recent seminal review paper underscored the reasons for the dismal progress in the field[3]. Ho and Huang have pointed out that before an effective vaccine can be devsied we must know about the speed of HIV-I growth in newly infected persons in whom viral titers rise exponentially even though they are yet asymptomatic. However very few studies are available regarding the same. The speed of HIV-I spread in the initial phase of the infecton is measured by the reproductive ratio R_o whch is the mean number of cells that become infected de novo by a single infected cell. Mathematically:[4,5,6]

$$R_o = \kappa\pi\lambda / cd\,\delta$$

Where

κ (infectivity constant) depends on the efficiency with which HIV-I infects a CD4 cell

π (Burst size is the number of virions produced by one infected cell

λ is the CD4 T cell production rate

c (Viron clearance rate constant) is the rate of viron clearance by the body.

d is the normal T cell death rate

δ is the death rate of productively infected T cells.

Accurate estimations of R_o are urgently needed as a vaccine can be only effective if it reduces R_o to a value <1 implying that the number of infected cells is decreasing with each successive generation. Ho and Huang have pointed out that CD4 cell production rate λ and normal T cell death rate d will not be affected by vaccination and hence vaccines can only reduce R_o by either decreasing k/c (humoral response) by producing antiibodies that neutralize HIV-I (decrease k) or opsonize HIV-I (increase c) or by decreasing π/δ by CD8 mediated responses (cell mediated response) which either suppress viral replication (decrease π) or cause death of infected cells (increase δ). Combination of both strategies will obviously have a multiplicative effect because $R \alpha \kappa/c$ x π/δ. Thus vaccines will be most effective only if they elicit both humoral and cell mediated responses.

Lowering k/c by vaccine induced humoral antibodies is problematic because variable sequences of sugars on gp 120 viral envelope glycoprotein shield the armoury of the virus with which it infects CD4 T cells (e.g. receptor (CD4) and Coreceptor (CCR5) binding sites) from attack by humoral antibodies hence killed whole virus vaccines and recombinant envelope proteins have little chance of success.[7,8,9] More tantalizing is the finding that monoclonal antibodies directed against critical regions of the virus like CD4 contact site (monoclonal antibody b12) or domains near receptor and coreceptor binding sites (antibodiy 2G12) can potently neutralize primary strains of HIV-I. However eliciting formation of such antibodies using envelopone immunogens is not yet possible. Modfied gp 120 from which elements shielding functional domains have been removed elicit more vigorous neutralizing antibody formation in animals. Huang and Ho suggest that similar efforts to modify gp41 envelope protein should be made as these must form a 6 bundle coiled coil structure before viral and Tcell membrane fusion can occur[10] and antibodies which prevent the fiormation of this coiled coil structure can prevent infection of T cells.[3]

The second approach of decreasing R_o by lowering π / δ ratio by inducing cell mediated responses is now attracting more interest because of the growing awareness about the importance of cytotoxic Tcells in controlling HIV-I infection. Live attenuated Simian Immunodeficiency Virus can promote virus specific cytotoxic T cell response in monkeys but use of live attenuated HIV-I vaccines in humans is dangerous and hence alternative strategies like DNA vaccines, and viral vectors like vaccina virus, canarypox virus, adenovirus, adenoassociated virus, alphavirus and vesicular stomatitis virus carrying one or more HIV-I genes are attracting more attention. A DNA vaccine and a recombinant adenovirus vaccine based on subtype A of HIV is undergoing phase I testing in England and Kenya. A DNA vaccine and a recombinant adenovirus

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vaccine based on subtype B are undergoing human trials in U.S. Several studies have shown that such vaccines can ameliorate disease progression and high levels of viremia in monkeys but do not prevent infection. Ho and Huang have stressed that because of high rate of mutation of HIV-1, vaccines should be directed against multiple viral antigens but most vaccines under development incorporate only one or two viral antigens which greatly reduces their chance of success.

There is another important lacuna in our knowledge referred to by Ho and Huang. The life span of a productively infected T cell is two days. On the first day (eclipse phase) no viral progeny is made and the cell dies the next day producing viral progeny in large numbers. Very little is known about the timing of expression of early and late gene products in the infected cells. We are also ignorant of the precise timing of recognition and destruction of infected T cells by cytotoxic T cells, yet these determne π and δ and knowing them is essential to the success of an approach based on lowering R_0 by reducing π/δ.

An additional hurdle in the development of an AIDS vaccine highlighted by Ho and Huang is that raised by the high mutation rate of the HIV-I genome. At present four subtypes or clades of HIV-1 namely A, B, C and D are known. Subtype B is dominant in U.S. and Europe while A, C and D have spread throughout Africa and subtype C is the most prevalent worldwide accounting for half of the new HIV-I infections worldwide. Most candidate vaccines are being developed by producers in U.S. and Europe and are based on subtype B but it is unlikely that countries in Asia and Africa with high HIV-1 prevalence rate of a different subtype (A, C or D) will allow their clinical testing which must needs be done in an area with a high HIV-1 prevalence rate. Ths must be taken into account in any program of HIV-1 vaccine design.

References:

1. Hajini GH, Iffat Hassan, Shah PA, Hussain ST. AIDS Vaccines - An Update. JK Practitioner 1996;4:2-5.
2. Nowak MA, McMichael AJ. How HIV defeats the immune system. Sci. Am. 1995;58-65.
3. David D Ho, Yaoxing Huang. The HIV-I Vaccine Race. Cell 2002; 110:135-138.
4. Nowak M, Lloyd A, Vasquez G et al. Viral dynamics of primary viremia and antiretroviral therapy in simian retrovirus infection. J. Virol 1997; 71:7518-7525.
5. Little SJ, Mclean AR, Spina CA et al. Viral dynamics of acute HIV-I infecton. J. Exp. Med 1999; 190:841-850.
6. Stafford MA, Corey L, Cao Y et al. Modelling plasma virus concentrations during primary HIV infection. J. Theor. Biol 2000;203:285-301.
7. Wyatt R, Sodoroski J. The HIV-1 envelope glycoproteins: fusogens, antigens and immunogens. Science 1998;280:1884-1888.
8. McMichael A, Hanke T. The quest for an AIDS vaccine: Is the CD8 + T Cell approach feasible. Nat. Rev. Immunol 2002;2:283-291.
9. Poignard P, Saphire EO, Parrenl PW et al. gp120: Biologic aspects of structural features. Ann. Rev. Immunol. 2001;19:253-274.
10. Chan DC, Kim PS. HIV entry and its inhibition. Cell 1998;93:681-684.

REMOVAL OF SURGICALLY ENTANGLED DRAIN: A NEW ORIGINAL NON-OPERATIVE TECHNIQUE

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***Abstract:** We devised a new original inon-operative technique of removing a surgically entangled drain by aseptic intralumenal introduction of Steinimiann pin/Kirschner wire and successfully used the method in vivo following bipolar hemiarthroplasty of hip. We also determined the "in vitro" reproducibility of the technique and the optimal diameter of Kirschner wires and Stenmann pins relative to the most common drains anid vicryl sutures. The most suitable wires were the thickest corresponding to the lumenal diameter. The trocar-pointed wires proved more effective. Thcker sutures through the middle third of the lumen were most easily felt. We found this technique simple and effective and could save the patient from re-operation.*

***Key words:** Method, Drain, Entanglement, Kirschner, Wire, Stenmann.*

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Introduction

Accidental suturing of the drain during closure of a surgical wound is not unusual and often results in re-operation. We successfully removed one such drain in a patient following bipolar hemiarthroplasty of hip, using a Steinmann pin to cut the offending suture through the lumen of the drain. We also tested our technique in vitor and achieved 100% success rate. No such technique for removal of surgical drains has been descrbed in literature.

Case Report

A sixty-eight year old man had bipolar hemiarthroplasty of right hp. A drain No. 14, on attempted removal 48 hours after surgery, was found accidentally sewn in soft tissues. Instead of subjecting the patient to re-operation, we decided to try our Steinmann pin technique in the ward under aseptic precautions. The drain and surrounding skin were prepared with Betadne surgical solution. The drain was cut near the skin with sterile scissors and its free end held with an artery forceps. Applying traction with the forceps, the drain was kept straight and taut to the point of holding stitch. A 2.5 mm trocar-pointed Steinmann pin was introduced through the lumen, holding stitch felt and cut (Figure-1) and drain removed easily. No haematoma, infection or muscle hernia resulted from the procedure.

Table-1: Optimal Kirschner wires/Steinmann pins corresponding to the size of the drains.

	Diameter of K-wires / Steinmann pins	
Size of drains	Introducible Diameters	Optimal Diameters
No. 10	0.45mm, 0.62mm	0.62mm
No. 14	0.45mm, 0.62mm, 0.80mm 1.0 mm, 2.0mm, 2.5mm	2.5mm
No 18	0.45mm, 0.62mm, 0.80mm 1.0mm, 2.0mm, 2.5mm, 3.2mm	3.2mm

Materials and Methods

We undertook to assess the "in vitro" reproducibility of the new method and determine the suitability of various Kirschner wires and Steinmann pins in relation to different drains and sutures.

Our measurements included the most commonly used suction drains (No 10, No. 14 and No. 18), vicryl sutures (No 3-0, No 2-0, No 1 and No. 2), Kirschner wires of diameters (0.45mm, 0.62mm and 0.80mm) and Steinmann pins of diameters (1mm, 2mm, 2.5mm and 3.2mm).

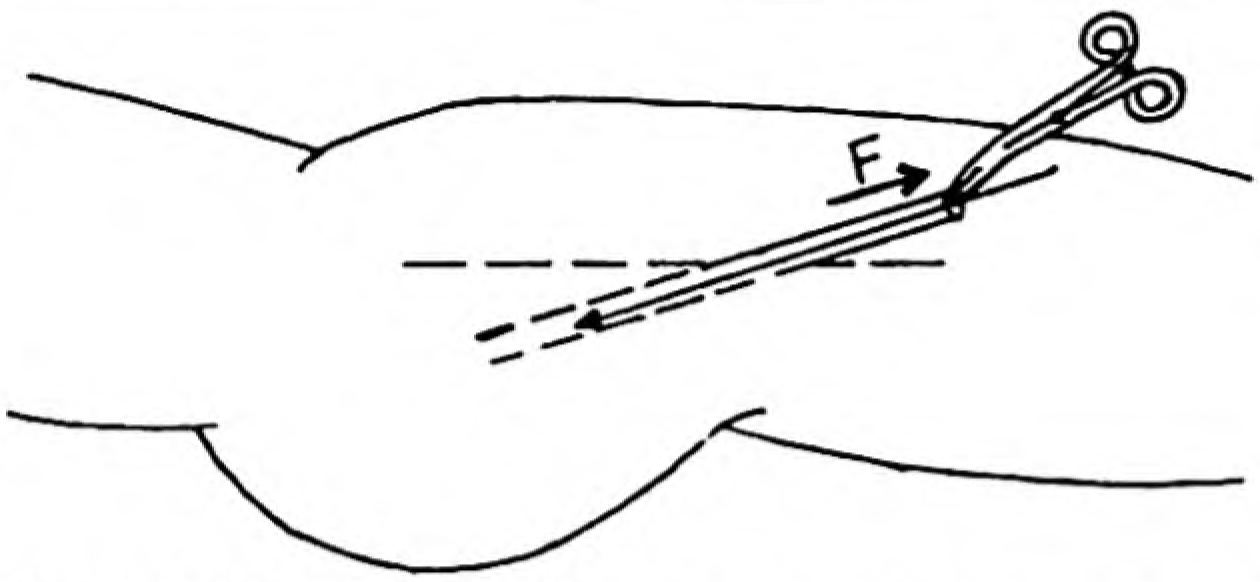

Figure -1: Diagram illustrating removal of drain by cutting the holding stitch using Steinmann pin. Note the drain is pulled and held straight under tension against the stitch.

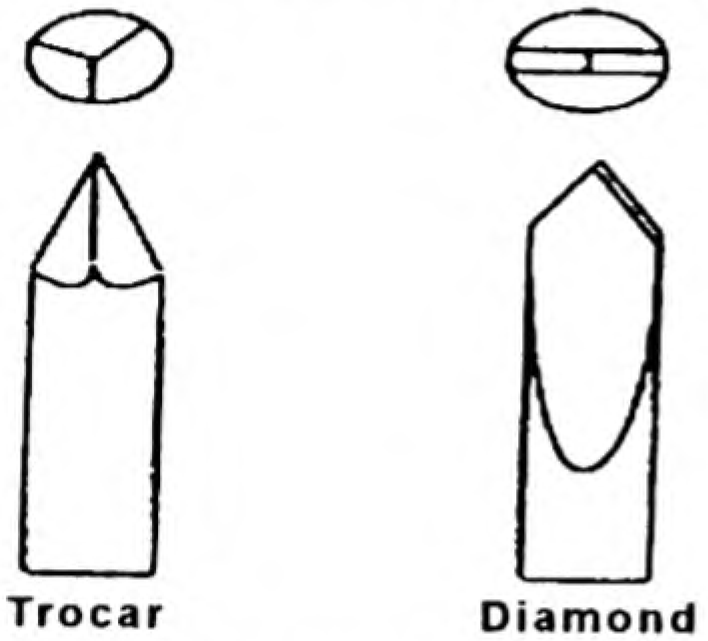

Figure-2: Diagram showing trocar and diamond pointed pins.

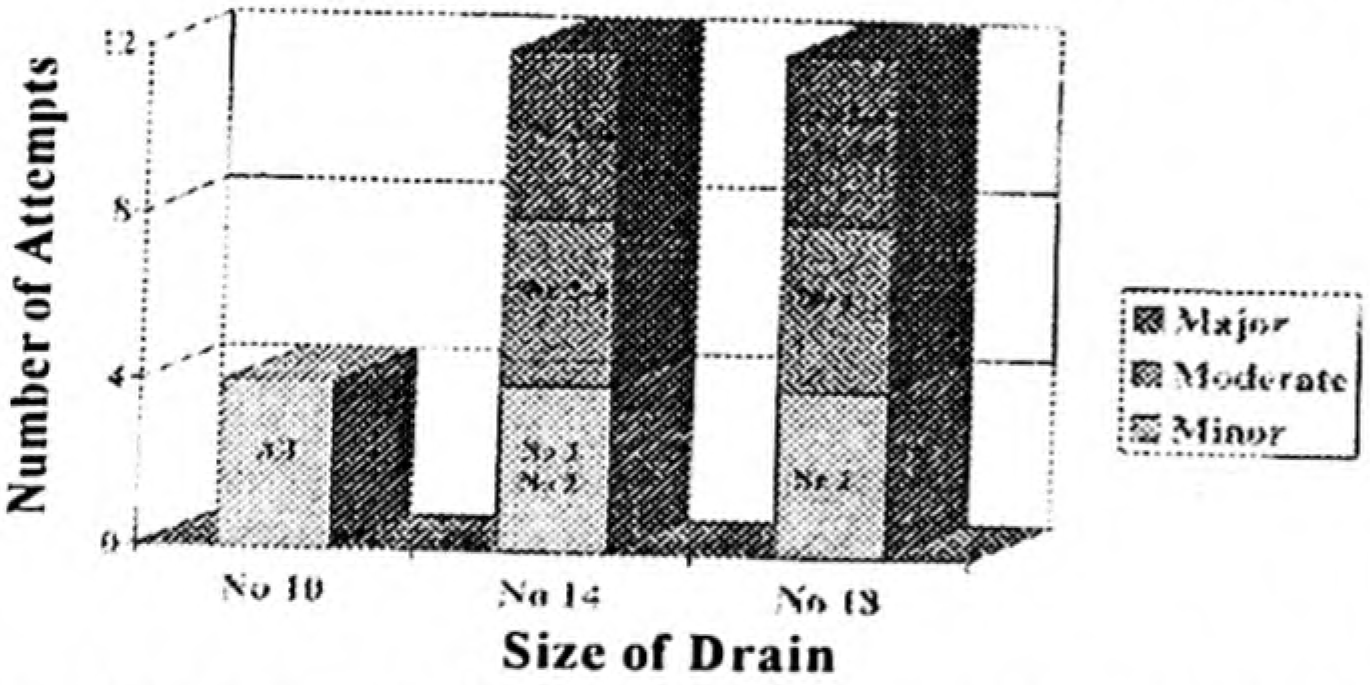

Figure-3: Degree of difficulty in cutting the suture with optimal wire, as defined by the number of attempts. Relation of the size of drain to the size of suture.

Trocar and diamond pointed Kirschner wires and Stenmann pins, after sharpening the cutting edges, were tested to assess suitability (Figure 2). The drain lumen was divided into thirds- superior, middle and inferior- to assess the degree of difficulty in finding the suture in different parts of the lumen and determine the most suitable wire or pin in each case. The level of difficulty was defined as minor, moderate and major according to the number of attempts for successful division of the stitch (Figure 3).

One end of the drain was fixed to a stable point and the other end held under tension maintaining the drain straight and taut. The "holding" stitch was also kept under constant tension. The Kirschner wire/Steinmann pin was then introduced through the lumen to feel and cut the stitch.

Results

The most suitable wires for cutting the stitch in any third of the lumen of draiins iNo. 10, 14 and 18 had diameters of 0.62mm, 2.5mm and 3.2mm respectively. These were the thickest introducible wires for each corresponding drain lumen. (Table 1).

Level of difficulty encountered in cutting every suture, using the most suitable wire for each drain was as follows: (Figure 3)

For drain No. 10, all sutures had minor difficulty.

For drain No. 14, vicryl No. 1 and No. 2 had minor, No 2-0 moderate and No 3-0 major difficulty. For drain No 18, Vicryl No. 2 had minor, No. 1 moderate and both No. 2-0 & 3-0 major difficulty.

Regarding the site of suture in the lumen, in No. 14 and No. 18 drains all sutures crossing the middle third iwere easily found and cut, while superior and inferior thirds proved difficult. In drain No. 10, vicryl sutures No 1 and No 2 were easily found and cut throughout the lumen, while No. 2-0 and No 3-0 were easily found only in the middle 1/3.

It was easier to locate the suture with the trocar-pointed rather than diamond tipped wires and a rotational motion during introduction facilitated easy cutting. The diamond pointed wires required their cutting edge perpendicular to suture line for successful removal of the stitch.

Although we experienced initial difficulty in locating the suture in early attempts, we soon developed the feel and technical ease to achieve a success rate of 100%.

Discussion

In the event of inadvertent surgical entanglement of a drain, we developed a new effective technque which saves the patient from re-operation by the simple expedient of cutting the offending suture with the "proper" sized trocar-pointed Kirschner wire or Steinmann pin, introduced through the lumen. We determined the optimal diameter of the Kirschner wires/Steinmann pins for each drain size- 0.62mm, 2mm or 2.5mm and 3.2mm for drains No. 10, No. 14 and No. 18 respectively.

The tissue plane, in which the drain is caught in the patent, can not be accurately reproduced "in vitro", but is not considered a significant factor for success as long as the drain is held straight under tension. Firm traction at the free end straightens the drain, facilitates introduction of the wire and minimises the risk of drain penetration. Furthermore, if the suture is around rather than through the drain, traction will pull the drain out automatically.

Conclusion

The technique of intralumenal introduction of Kirschner wire or Steinmann pin to feel and cut the offending suture and facilitate non-surgical retrieval of a surgically entangled drain s a novel concept and saves the patient from re-operation. With due care and strict aseptic precautions, risk of infection should be no higher than operative removal of the drain.

POLYCYSTIC DISEASE OFTHE LIVER AND KIDNEY

A 60 year old man admitted as a case of polycystic liver and kidney disease with renal failure. Ultrasonography and MRI abdomen shown alongside.

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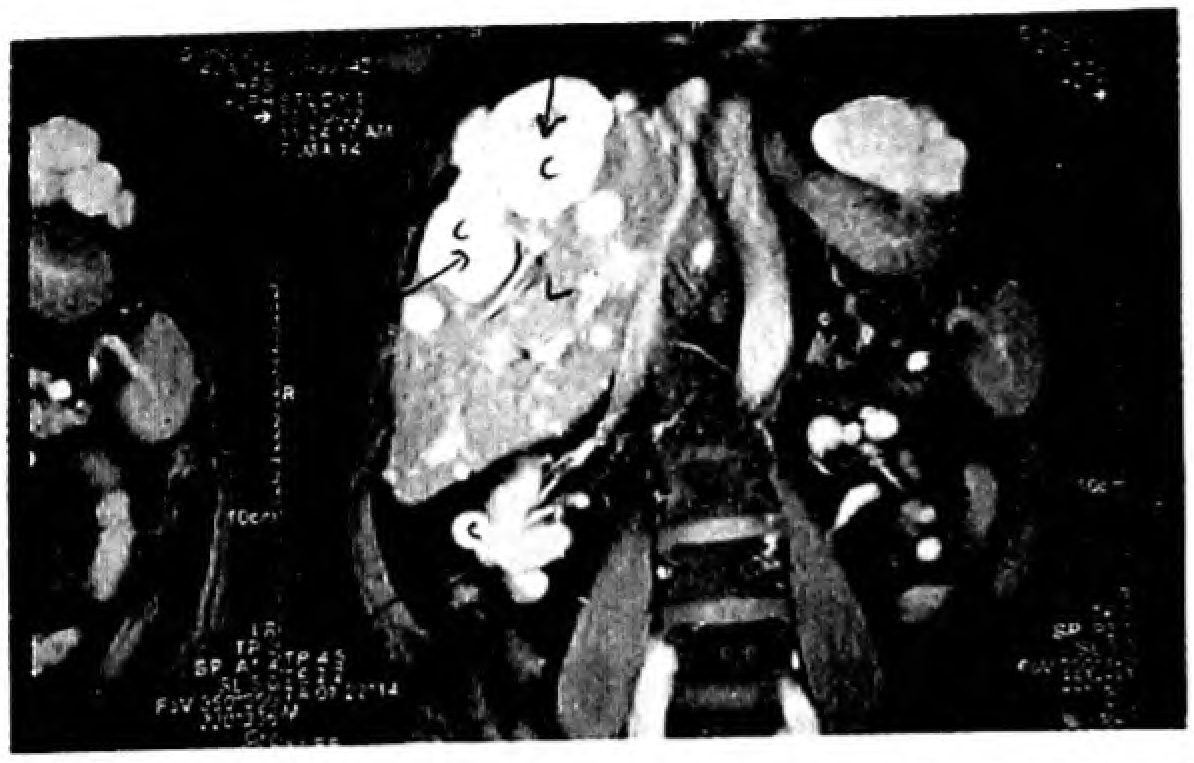

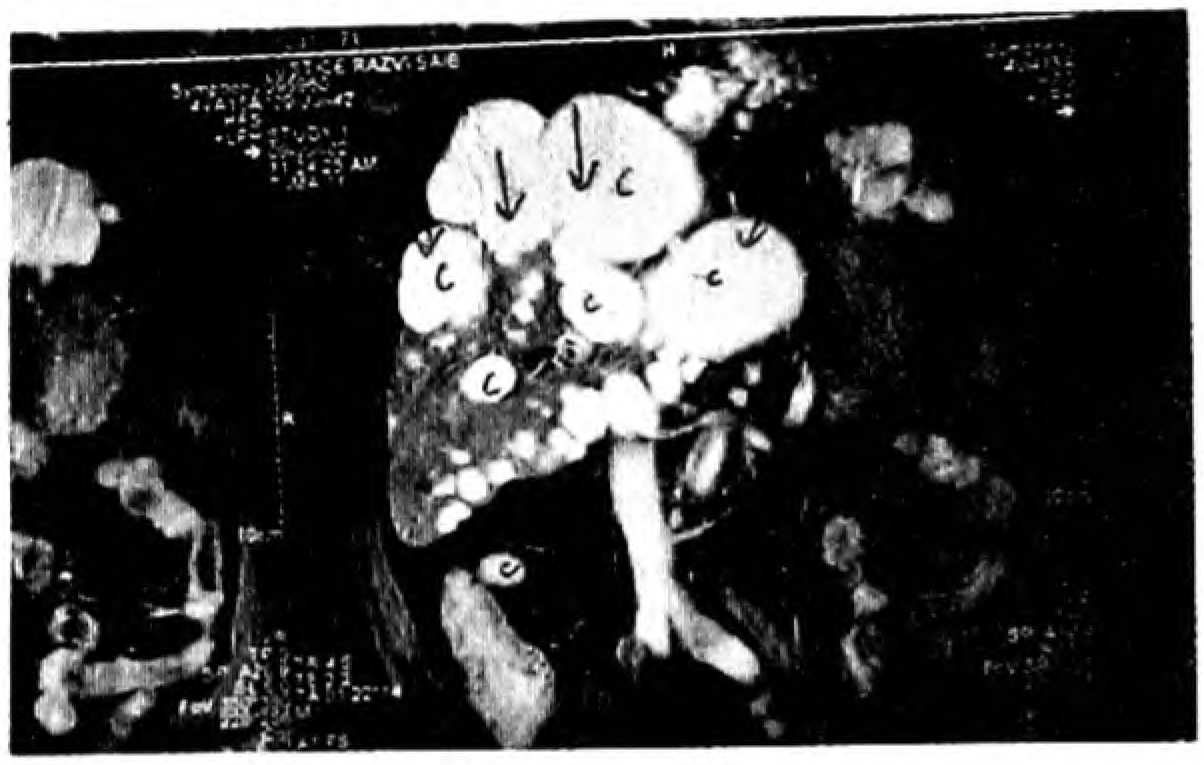

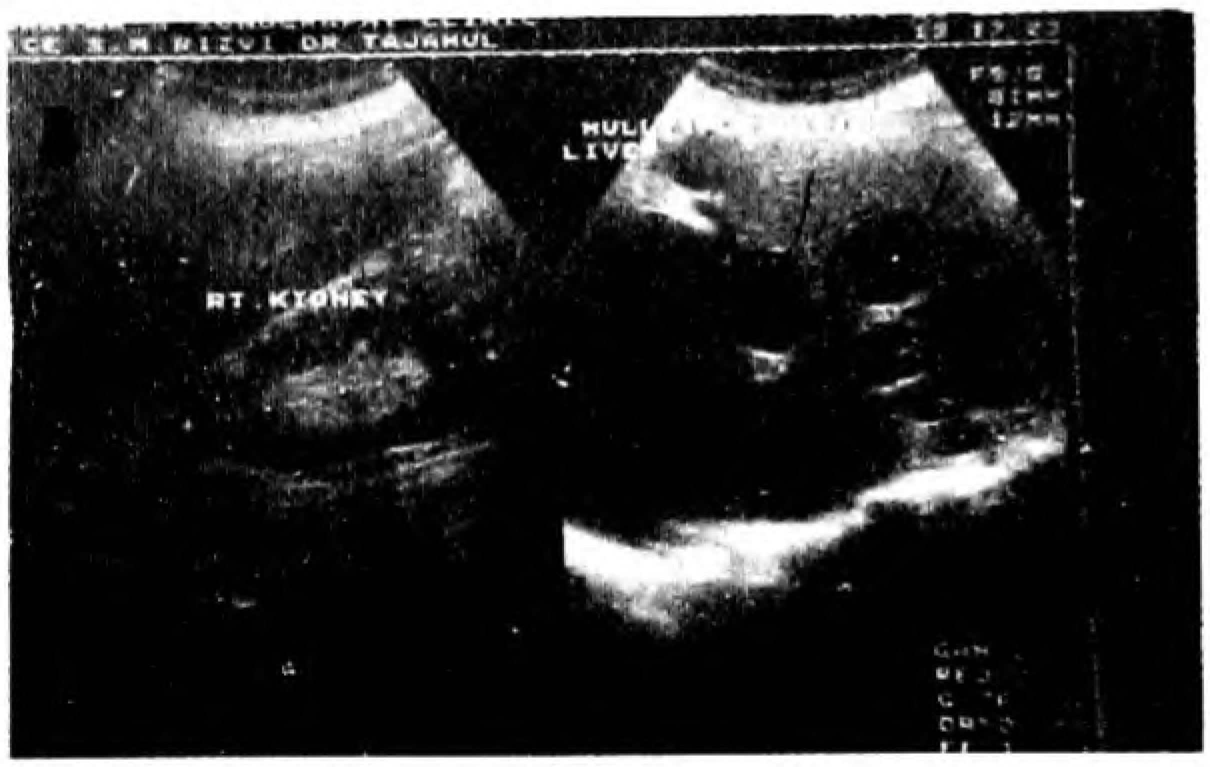

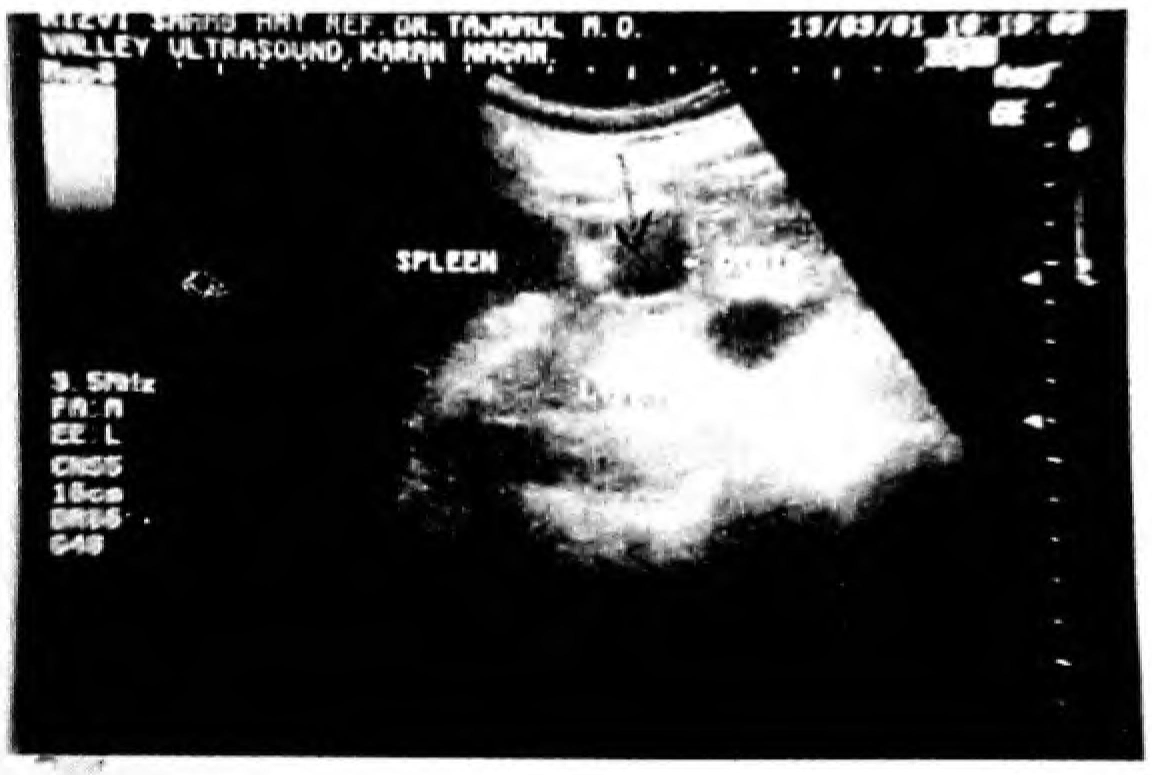

Fig.1: MRI Abdomen Coronal Section Liver is enlarged Multiple rounded hyperintense areas of variable sizes are seen in liver and kidneys and small cysts in peritoneal cavity.

Fig.2: MRI Abdomen. Coronal section. Liver is enlarged Multiple rounded hyper intense areas seen in liver and peritoneal cavity. Image also shows abdominal aorta till its bifurcation

Fig.3&4: USG Abdomen. There are multiple Anecholic areas of variable sizes in Liver / Kidney, consistent with polycystic disease of Liver / Kidney.

ORTHOPAEDICS:- MEDIVEAL TO MODERN

Suhail Afzal M.S.(Ortho); Mohammad Ramzan Mir M.S. (Ortho)

INTRODUCTON.

Orthopaedics ,like many specialities, has developed through a necessity. A necessity to correct deformity, restore function and alleviate pain. Orthopaedic surgeons have developed an ability to prevent major losses of bodily function and indeed they can prevent otherwise inevitable death. They seek perfection of their art, by ensuring that the patient reaches optimal condition in the shortest period of time by the safest possible method.

History is very important to any surgeon, particularly the Orthopaedic surgeon. The orthopaedic surgeon has once again been presented with advancing technology. This technology must be applied to the surgeon's practice, but it is best applied only when the surgeon has an underlying knowledge of the history of his art. He must be aware of the way surgeons in the past have contributed to orthopaedics and more importantly, of the mistakes that they have made in the process. The surgeon who makes a mistake that has been made by someone before him, is surely humbled and seen as poorly educated. So is he who states that he has developed a technique that no one has thought of before, because chances are that it has been thought of in the past.

In order for orthopaedics to advance in an optimal manner, it is clear that attention must be paid to history of Orthopaedics. The past is our foundation for future developments, we must build upon it so that we too can act as a stable foundation for future generations.

ANCIENT ORTHOPAEDICS:-
PRIMITIVE MAN:

Although we have no written historical accounts, primitive man provides us his fossils. These show that the same pathology affecting bone existed in primitive times. Evidence of fractured bones has been found, in some of which union has occurred in very fair alignment. This is interesting to note, as it gives us an ethical manner in which we can see the effects of no treatment at all,i.e. applying rest by instinct and early motion. It is inevitable that, at some stage, primitive man created a very crude splint, and that from that stage on, its advantages were recognized. Primitive man was probably also the first to perform crude amputations, and to trephine the skull.

ANCIENT EGYPT:

Mummified bodies, wall paintings and hieroglyphics, have shown us that people of the Egyptian age suffered from the same problems that we suffer today. They also show us some of the orthopaedic practices of that time. Splints have been found on mummies and they were made of bamboo, reeds, wood or bark, padded with linen. There is also evidence of the use of crutches, with the earliest known record coming from a carving made in 2830 BC on the entrance of a portal on Hirkouf's tomb.

Perhaps the most important source describing the practices of Ancient Egyptians comes from a papyrus, which was stolen from a tomb in 1862. The papyrus was then sold to an American Egyptologist by the name of Edwin Smith. The author is believed to be Imhotep. In this papyrus, injuries were clasiified according to their prognosis into three categories: an ailment which they would treat, an ailment that they would contend and an ailment which they would not treat. Discharge was reffered to as "ryt" , this is presumably the pus of osteomyelitis.

ANCIENT GREECE:

Many principles behind conditions and their treatment have been attributed to the Ancient Greeks. They could be regarded as the first to use a scientific approach, however they were also the first to document in detail their history and developments.Homer alone, in his account of the Trojan war, has provided us with an adequate insight to the understanding of injuries at that time and the treatment used for those injuries. The Iliad also contains references to various deformities. The Greek anatomists of Alexandria, during the 3rd century BC were also great contributors. Herophilus, who is believed to have practised human dissection, is regarded as the first to divide nerves into sensory and motor components and also the first to distinguish arteries from veins. Hegetor also of Alexandria, but of 100 BC, described in detail the anatomical relations of the hip joint, and was the first to record a description of the ligamentum teres.

In the period between 430 and 330 BC a very important Greek text was collated and is known as the Corpus Hippocrates. It is named after Hippocrates who is known as the father of Medicine. Hippocrates was born on the

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island of Cos in 460 BC and died at an old age in 370 BC. He is known as having brought a systematic and scientific approach to Medicine and as having defined for the first time the position and the role of a doctor in society. Although centuries have passed, the Hippocratic oath will always remain central to our practices.

Various volumes in the Corpus Hippocrates had relevance to Orthopaedics. One such volume is the one on joints. Here dislocation of the shoulder was described together with the various methods used in reduction. There were also sections describing the reduction of acromioclavicular, temporomandibular, knee, and hip and elbow joint dislocations. The correction of club foot was described. The problem of infection after compound fractures was described and treated with pitch cerate and wine compresses without forcible bandaging. Probing into any compound fracture was avoided.

Hippocrates had a thorough understanding of fractures. He knew of the principles of traction and counter-traction. He developed special splints for fractures of the tibia, similar to external fixation. Hippocrates also developed the Hippocratic bench or "scamnum". Of all the developments that Hippocrates has given to us, his careful clinical observation and rationale thinking must be particularly commended.

THE ROMAN ERA:

Althogh the teachings of Hippocrates were to dominate thinking for many centuries after his death, there are several contributors to Orthopaedics worthy of mention. During the Roman era, there was another respected Greek figure by the name of Galen (129-199 BC). He was originally from Pergamon and became a gladiatorial surgeon there before travelling to Rome. Galen is often referred to as "the father of sports medicine". He gave a good account of the skeleton and the muscles that move it. In particular, the way that signals are given from the brain through the nerves and to the muscles. He first recorded a case of cervical ribs. He described bone destruction, sequestration and regeneration in osteomyelitis and sometimes performed resection in such cases. Galen is believed to be the first to have used the Greek words, kyphosis, lordosis and scoliosis for the deformities described in the Hippocratic texts. He also devised several methods for correcting these deformities.

During this Graeco-Roman period, there were also attempts to provide artificial prostheses. There are accounts of wooden legs, iron hands and artificial feet. Soranus of Ephesus is said to have first described rickets. Ruphus of Ephesus described tendon ganglia and their treatment by compression. Antyllus of the 3rd century is said to have practised subcutaneous tenotomy to relieve contractions around a joint. It is said that he used both linen and catgut sutures for thee procedures. Various drills, saws and chisels were also developed during this period.

THE ARAB ERA:

Another Another Greek named Paul of Aegina (625-690 A.D.) worked in Alexandria and wrote "The Epitome of Medicine" which consisted of seven books based on the Hippocratic texts. The sixth book dealt with fractures and dislocations. With the invasion of Alexandria by the Moslems, many great books such as these were taken and translated into the Arab language. The great library of Alexandria was burnt. Although the Arab practices were regarded as an extension of those of the Greeks, the use of plaster-of Paris in the 10th century was significant. With the addition of water to a powder of anhydrous calcium sulphate a hard crystalline material was produced. A Persian by the name of Abu Mansur Muwaffak described the coating of plaster for fractures and other bony injuries of the limb.

THE FOUNDATION OF MODERN ORTHOPAEDICS:

It was not until the 12th century that Europe began to awake gradually from its Dark Ages. Universities and hospitals were beginning to be established, human dissection resumed and the great Greek texts were being translated from Arabic to Latin. However, until the 16th century, all developments remained within the shadow cast by Hippocrates.

AMBROISE PARE (1510-1590)

Ambroise Pare is regarded as the most famous surgical figure of the 16th century and the father of French Surgery. He was born in Bourg Herent in France. In 1532 he became an apprentice to a Parisian barber-surgeon, then worked for four years at Hotel Dieu in Paris. In 1541, he became a master barber-surgeon and did some work as an army surgeon. In 1564, he published a monumental work on Surgery, the Dix Livres de la Chirurgie. The first part contained Anatomy and Physiology and the second, Surgery. In this, many surgical techniques were described, one of the most significant being the use of ligature for large vessels in amputations. He also used a tourniquet in his amputations, to hold the muscles retracted with the skin, prohibit the flux of blood and to dull the senses. He designed a wide variety of forceps, instruments and braces of all kinds. With the help of armourers, he made a variety of artificial limbs from iron. The majority were cosmetic, although Pare did design a scoliosis corset and a clubfoot boot.

NICHOLAS ANDRY (1658-1759)

Andry was the professor of Medicine at the University of Paris and Dean of the faculty of Physick. In 1741, at the age of 81, he published a famous book called Orthopaedia: or the Art of Correcting and Preventing Deformities in Children. By such means that may easily be put into Correcting and Preventing Deformities in Children. By such means that may easily be put into practice by parents themselves and all such as are employed in Educating Children. In this book, Andry presents the word Orthopaedic, which derives from the Greek words straight and child. Andry was interested in postural defects and this has been reflected by his famous illustration, which is known The tree of Andry. Andry believed that skeletal deformities were due to faults of posture and shortness of muscles. Some regard Andry as the Father of Orthopaedics, by many strongly disagree, believing that his work was un-scientific and that his only contribution was the use of the word Orthopaedics.

THOMAS SYDENHAM (1624-1689).

Sydenham is likened to Hippocrates because his writings cover a large field and are characterised by good observation. Likewise, he is also known as the father of English medicine. He was born at Winford Eagle, and studied at Oxford and Montpellier. He himself suffered from gout and wrote an excellent description of the disease, detailing the attack, the changes in urine and the link with renal stones. He described acute rheumatism, chorea, and the articular manifestations of scurvy and dysentery.

PERCIVAL POTT (1714-1788)

Pott was from London and worked in St. Bartholomew's Hospital, were he received the diploma of the Barber-Surgeons' Company in 1763. He is best known for the fracture that bears his name Pott's fracture, as he was the first to give a good description of this ankle fracture. In 1756, he received a fracture of his own. This was an oblique compound fracture of the lower third of the tibia, which was acquired after falling from his horse. He refused to be moved until he had purchased a door to be carried on, as he believed that the jolting of a carriage would have exacerbated the injury. Immediate amputation was usually conducted on such injuries, but at the last moment amputation was stopped and the limb was saved. Pott's most famous work is on the paraplegia of spinal tuberculosis, where he stressed that the condition was not related to spinal cord compression, but associated with strumous disorders in the lungs. This is known as Pott's paraplegia.

WILLIAM HEBERDEN (1710-1801)

Heberden was born in London were he also built up a busy practice. He is known for initiating the Medical Transactions in 1766, but even more so for his description of Heberden's nodes.

JOHN HUNTER(1728-1793)

Hunter worked on a Lowland farm until he was 20 years of age. Until he was 32, he was a pupil and house surgeon at St. George's Hospital in London and also worked in his brother's dissecting room in Covent Garden. In the Seven Years' War, he served as a military surgeon. He set up a research centre in London's Golden Square and taught and lectured at Leicester Square until angina eventually led to his death. Hunter's contribution was immense and even stemmed through the pupils he taught (e.g. Abernethy, Chessher, Jenner and Philip Syng Physick). Hunter himself was a pupil of Percival Pott. Although he received little formal education (unlike his brother William, an obstetrician in London) Hunter put the practice of surgery on a scientific foundation and laid the framework for the twentieth century developments. His saying Don't Think, try the experiment has inspired generations of modern surgeons.

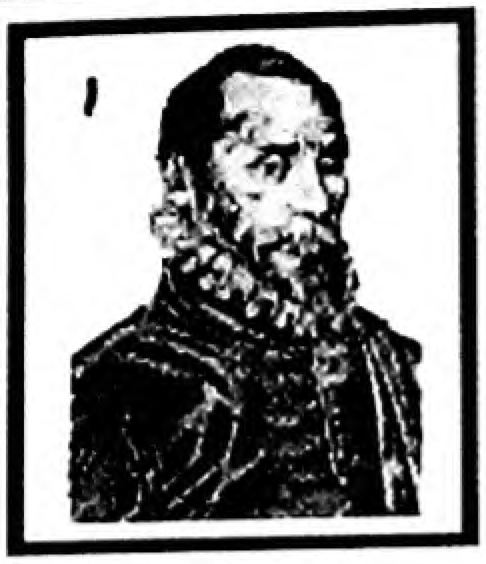
Much of Hunter's knowledge may be attributed to his military experience and his experiments on animals. He described how to assess muscle power in a weak muscle. With joint injury and disease, he states that voluntary movement should not be permitted until inflammation has settled, otherwise contracture is promoted. He believed that healing depended on the body's innate power, and that the surgeon's task was to aid this. Hunter believed that bone disease often required mechanical assistance. He studied loose bodies in joints, pseudoarthroses and fracture healing, where he described the transformation from fracture haematoma to fibrocartilagenous callus to the deposition of new bone, trabeculation, reestablishment of the medullary canal and the resorption of excess bony tissue. Hunter wrote A Treatise on the Blood, Inflammation and Gunshot Wounds in 1794, and also made attempts at tissue grafting.

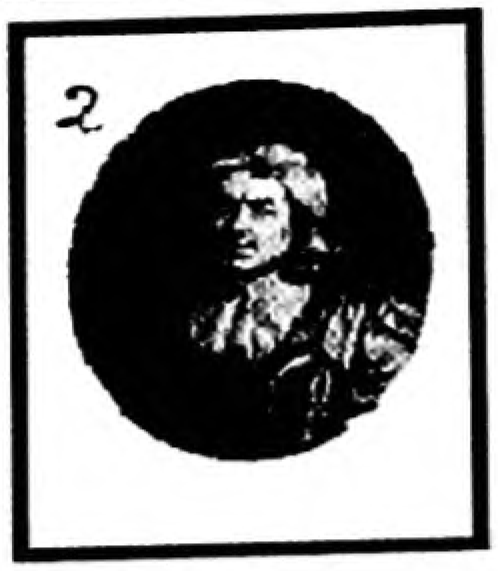
His collection of specimens (initially over 14,000 POT's; half destroyed in the bombing of London) is in the College of Surgeons, London. They describe the development of the various systems from the simplest (insects) to the most complex. It is a humbling and inspiring experience to visit the museum and see one mans monumental contribution to surgery.

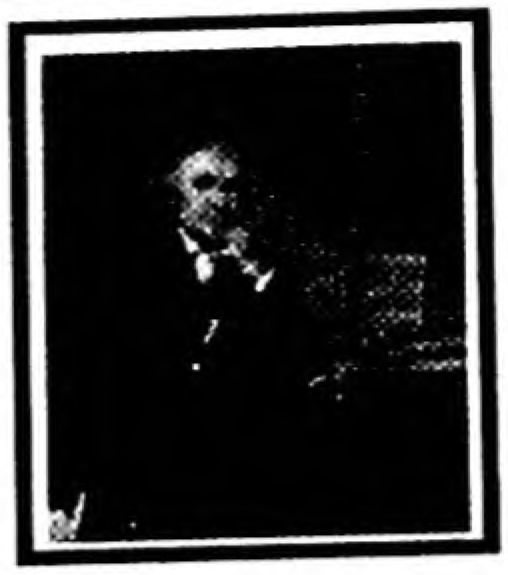
JEAN-ANDRE VENEL (1740-1791)

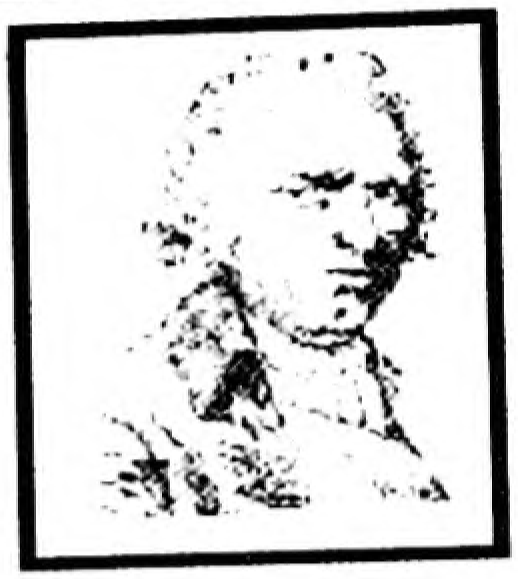
Jean-Andre Venel was a Genevese physician who studied dissection at Montpellier at the age of 39, and in 1780, established the first orthopaedic institute in the world at Orbe, in Canton Waadt.

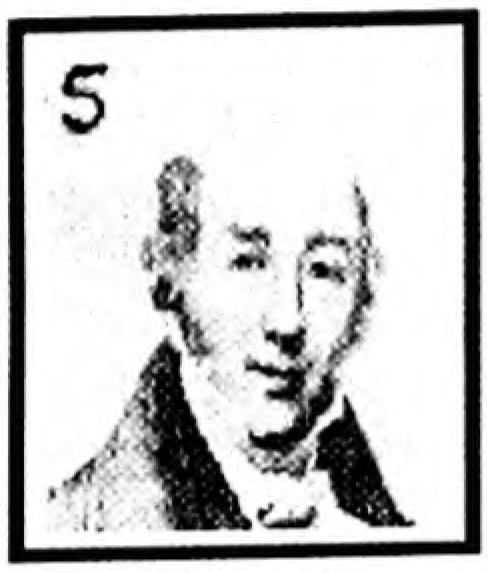
This was the first true hospital that dealt specifically with the treatment for crippled children's skeletal deformities. Venel recorded and published all his methods and for this was known as the first true orthopaedist. He is also regarded as the father of orthopaedics, as his institute acted as a model for hospitals throughout Europe. Venel

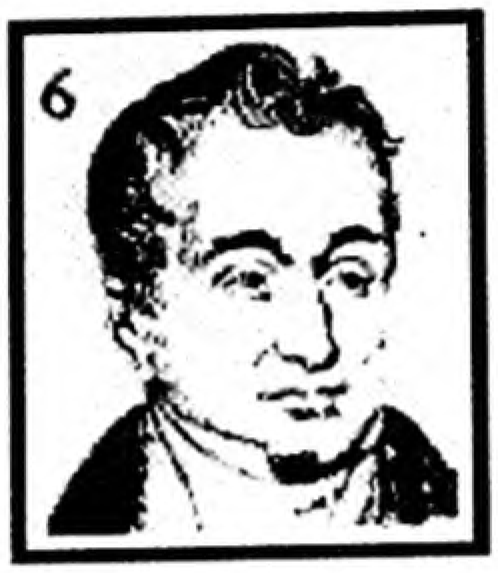


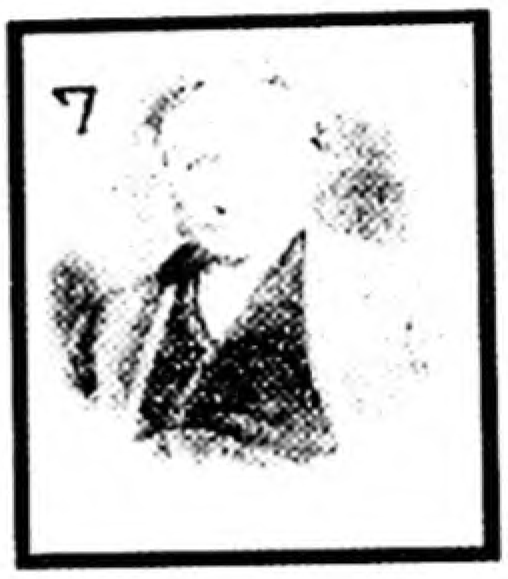


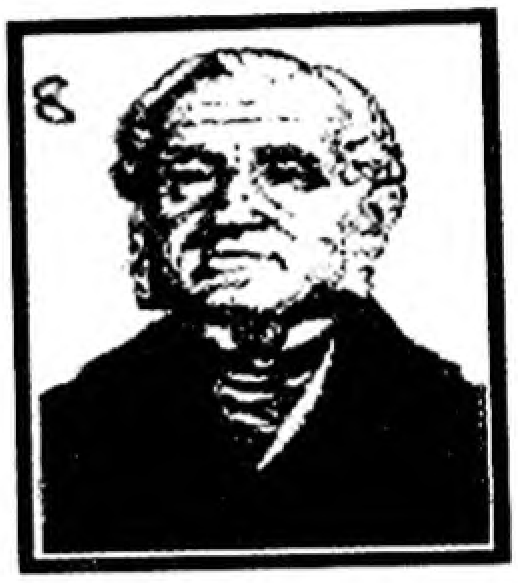


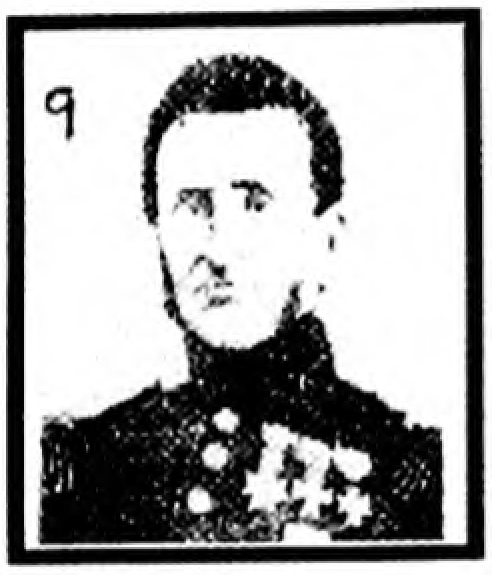


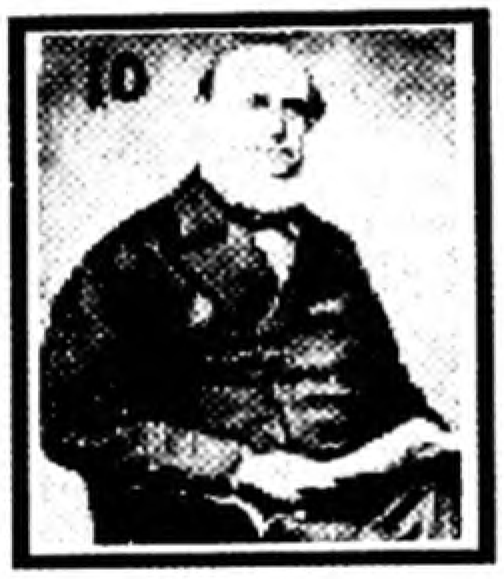


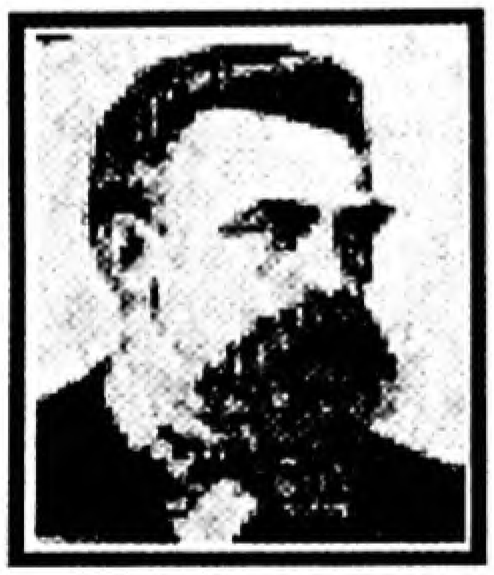


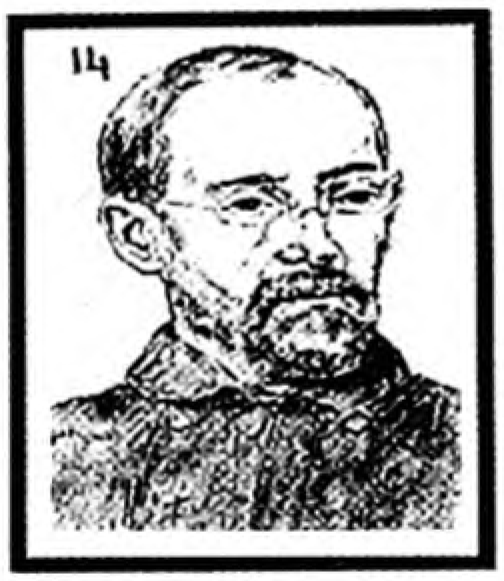


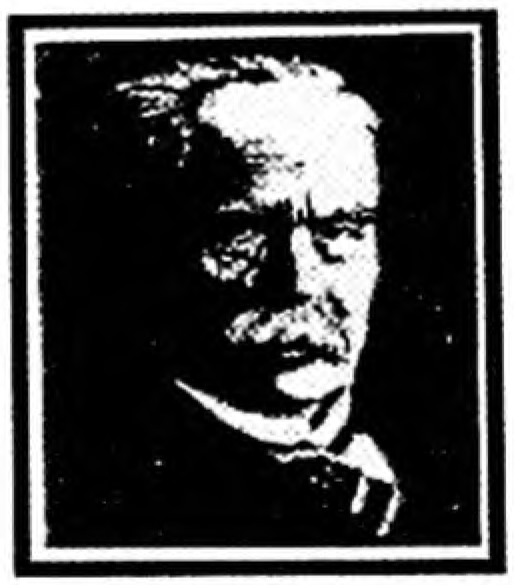


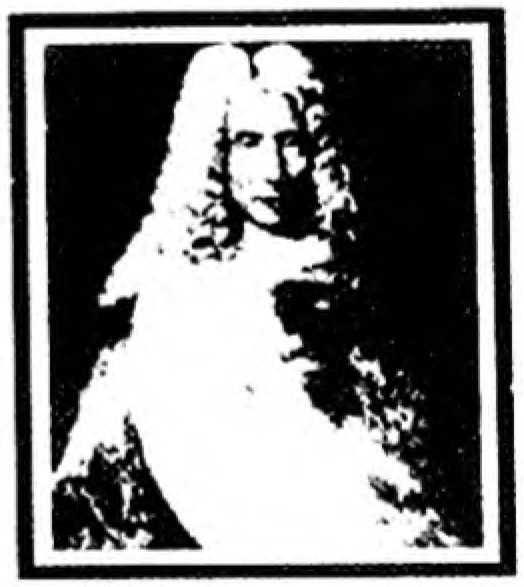


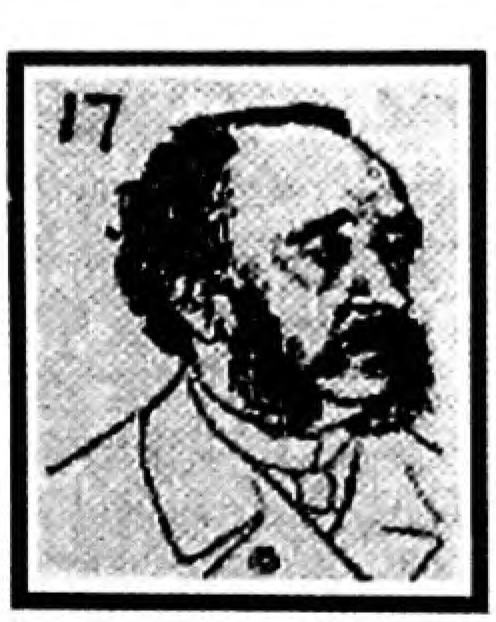












1. AMBROISE PARE (1510-1590). 2. THOMAS SYDENHAM (1624-1689). 3. WILLIAM HEBERDEN (1710-1801). 4. JOHN HUNTER (1728-1793). 5. ABRAHAM COLLES (1173-1843). 6. BARON GUILLAUME DUPUYTREN (1777-1835). 7. JAMES SYME (1799-1870). 8. SIR BENJAMIN BRODIE (1786-1862). 9. ANTONIUS MATHYSEN (1805-1878). 10. WILLIAM JOHN LITTLE (1810-1894). 11. SIR JAMES PAGET (1814-1899). 12. RICHARD VON VOLKMANN (1810-1894). 13. HAROLD BENNETT (1837-1907). 14. HUGH THOMAS (1834-1891). 15. FRITE DE QUERVAIN (1868-1940). 16 NICHOLAS ANDRY(1658-1759). 17. FRIEDRICH TRENDELENBURG(1844-1924)

stressed the importance of sunlight and made various braces and appliances at the workshops within the institute.

WILLIAM HEY (1736-1819)

William Hey was born in Pudsey near Leeds. At the age of 14 he was apprenticed to a surgeon and apothecary and nearly died of an overdose of opium whilst studying its effects. He was the founder of Surgery at Leeds and trained at St. George's Hospital. Hey wrote a book on Surgery which contained several chapters on Orthopaedics. Subacute Osteomyelitis of the tibia was described and he advocated deroofing of the lesion. In 1773, Hey banged his knee getting out of the bath, and many attribute his subsequent interest in the knee to this. He coined the phrase internal derangement of the knee, and described meniscal injuries. Hey described loose bodies and introduced tarso-metatarsal amputation.

GIOVANNI BATTISTA MONTEGGIA (1762-1815)

Monteggia was born at Lake Maggiore and was a Milanese pathologist who acquired syphilis by cutting himself at autopsy and became a surgeon and professor at Milan. He is particularly remembered for his description in 1814 of the fracture that bears his name, Monteggia's fracture.

ABRAHAM COLLES (1173-1843)

Colles was born in Kilkenny, Ireland, of humble origins. Nevertheless, he became professor of Surgery at the College of Surgeons in Dublin from the age of 29. He was the first to tie the subclavian artery, but is best known for his description of Colles' fracture, in 1814 (the same year as Monteggia.

BARON GUILLAUME DUPUYTREN (1777-1835)

Was born in central France. He was kidnapped as a boy by a rich person from Toulose on account of his good looks. He was taken to Paris and endured great poverty during his studies. Dupuytren became surgeon in chief at the hotel Dieu and worked tremendously hard and became rich. He was described as an unpleasant person to meet, yet his work was wonderful to read. He was characterized as Firest among surgeons, Last among an accurate clinical observer with agreat interest in pathology. He is most asscociated with the contracture of palmar fascia and ankle fracture that he described. He wrote on many subjects, congenital dislocation of hip, the nature of callus formation, exostosis, the trendelenburg sign, tenotomy in torticollis.

JAMES SYME (1799-1870)

Syme was born in Edinburgh. As a student at Edinburgh University he found a way of dissolving rubber. Syme opened a school of Anatomy and later opened a very successful private clinic. In 1833, he became Professor of Surgery in Edinburgh and held that position until his death. (he had actually made an agreement with his predecessor to pay him a pension if he resigned). Syme is known for introducing conservative alternatives to the major amputations that were carried out at the time. In 1831, he released a booklet, which detailed cases where joint excision could be used instead of amputation for grossly diseased joints, as in tuberculosis, and injured joints. In 1842, Syme described an amputation at the ankle. This amputation bears his name, as it replaced a portion of below knee amputations, which were ordinary practice at that time.

SIR BENJAMIN BRODIE (1786-1862)

Brodie was a national figure. He was a surgeon at St. George's Hospital and a friend of the Thomas family (that of Hugh Owen Thomas). He first published his book, On the Diseases of Joints in 1819, which proved to be a popular reference for many years. In 1832, he described the chronic bone abscess that has been named after him. The patient was a man of 24 who had recurring symptoms in the lower extremity of his right tibia. On examination, Brodie found a pus filled cavity, for which he believed that amputation could be avoided by trephination of that cavity. He recognised the association of arthritis with gonorrhoea and that all children's hip disorders were associated with infection. IN 1843, he introduced the Fellowship examination of the Royal College of Surgeons in order to improve the education and standing of surgeons.

JOHN RHEA BARTON (1794-1871)

Barton was born in Lancaster, Pennsylvania, U.S.A. He studied at the Pennsylvania Hospital and later worked for Physick (the father of American Surgery) who in turn was a student of Hunter's.It was said that Barton was ambidextrous and that once he had positioned himself for an operation, he did not move about. In 1826, he performed a subtrochanteric osteotomy of the femur for a severe flexion-adduction deformity of the hip. Barton is best known for his innovative corrective osteotomies for ankylosed joints. In 1834, Barton wired a fractured patella and in 1835, he described Barton's fracture of the wrist.

ROBERT WILLIAM SMITH (1807-1873)

Smith was born in Dublin, he studied and worked there. He became Professor of Surgery at Trinity College in Dublin. Smith founded the Dublin Pathological Society with Colles, Graves, Corrigan and Stokes. In 1847, Smith wrote a classic book called A Treatise on Fractures in the Vicinity of Joints, and on certain forms of Accidents and Congenital Dislocations. Here he describes the eponymous Smith's fracture, and Madelung's deformity before Madelung described it. In 1849, he published A Treatise on the

Pathology, Diagnosis and Treatment of Neuroma. This book was said to be so large that it was larger than an ordinary sized dinning-room table when opened up. Smith wrote on neurofibromatosis in great detail, much before von Recklinghausen did.

ANTONIUS MATHYSEN (1805-1878)

Mathysen was a Dutch military surgeon who in 1851, invented the plaster of Paris (POP) bandage which was to become so important to orthopaedic practice.To this day, a POP cast is the mainstay of fracture immobilisation.

WILLIAM JOHN LITTLE (1810-1894)

Little was educated at the Jesuit seminary at St. Omer. He himself had a paralytic clubfoot. The treatment in London was amputation, however, he found a cure in Germany by tenotomy. Little was a founder of the Royal Orthopaedic Hospital. He published a detailed report, in 1862, of the then ill-understood group of deformed and partly retarded children and young adults. This type of spastic paralysis with paraplegia of the lower limbs was then called Little's disease for many years.

JOSEPH LISTER (1827-1912)

Lister studied at the University College Hospital in London. In 1853, he became Syme's house surgeon, he then married Syme's daughter, and in 1854 became assistant surgeon to the Royal Infirmary. Lister is known for the introduction of antisepsis. He first applied carbolic acid to a compound fracture in 1965. It was soon clear that the practices had had a dramatic effect in reducing in particular abscesses, pyaemia, hospital gangrene, erysipelas and amputation mortality. Lister was made a baronet in 1883, and later in his life was thought to have trialed the application of the Penicillium mould directly to wounds.

JEAN-MARTIN CHARCOT (1825-1893)

Charcot was from Salpetriere in Paris and is known worldwide as the first professor of Neurology. He wrote a thesis distinguishing gout, rheumatoid arthritis and osteoarthritis. Charcot also first described the arthropathy that bears his name. Charcot's joints. He was first to write about amyotrophic lateral sclerosis, intermittent claudication, disseminated sclerosis, intermittent hepatic fever and herpes zoster.

THEODOR KOCHER (1841-1917)

Kocher was born in Berne and studied in Berlin, London, Paris and Vienna. In 1872, he became Professor of Surgery in Berne. Kocher had a great interest in Anatomy and in 1870, he described his eponymous method of reducing a dislocated shoulder. Kocher wrote a remarkable book in which he detailed many useful surgical incisions that he had developed, such as his posterolateral exposure of the hip. He also developed several surgical instruments, but his main interest was in Kocher was born in Berne and studied in Berlin, London, Paris and Vienna. In 1872, he became Professor of Surgery in Berne. Kocher had a great interest in Anatomy and in 1870, he described his eponymous method of reducing a dislocated shoulder. Kocher wrote a remarkable book in which he detailed many useful surgical incisions that he had developed, such as his posterolateral exposure of the hip. He also developed several surgical instruments, but his main interest was in thyroid disease. Kocher was born in Berne and studied in Berlin, London, Paris and Vienna. In 1872, he became Professor of Surgery in Berne. Kocher had a great interest in Anatomy and in 1870, he described his eponymous method of reducing a dislocated shoulder.

SIR JAMES PAGET (1814-1899)

Paget was a graduate of St. Bartholomew's Hospital in London, where he remained for the rest of his career. It was in 1877 that Paget gave the first description of what he called osteitis deformans, but what is now commonly called Paget's disease. He noted the increased incidence of osteosarcoma, the increasing head size and deformities. (One of Paget's original drawings is shown.) Paget was also a remarkable lecturer with a great interest in bone pathology. His name is also associated with other pathological processes.

SIR WILLIAM MACEWEN (1848-1924)

Macewen studied in Glasgow and had Syme and Lister as teachers. The new era of antisepsis enabled him to make many contributions to Surgery. In terms of his Orthopaedic contributions, he performed many osteotomies and developed a one-piece osteotome. Macewen's main research interest was in bone growth and in 1879 he performed the first of his pioneering bone grafts. Many of his grafts were performed on people who had had portions of their bones excised, but who had otherwise normal function. Macewen was also a pioneering neurosurgeon and cardiothoracic surgeon. He worked on cerebral tumours and abscesses and also performed the first pneumonectomy.

RICHARD VON VOLKMANN (1830-1889)

Volkmann was from Halle, Saxony. He was the first in Germany to institute Lister's antiseptic methods. In 1881, Volkmann published his famous paper on ischaemic muscular paralyses and contractures. Here he attributed the cause of the contractures to direct changes in the muscles produced by arterial occlusion and emphasised the early warning of preliminary weakness. These contractures are otherwise know as Volkmann's ischaemic contractures. It is interesting to note that Volkmann wrote popular poems and fairy stories and also founded a surgical journal.

EDUARD ALBERT (1841-1900)

Albert was born in Bohemia and studied in Vienna. He is best known for producing artificial ankyloses in paralysed limbs and wrote a paper on this in 1881. Albert performed tarsal and shoulder arthrodesis for paralysis and recurrent dislocation, and was the first to use the term arthrodesis. Albert also described synovectomy, the transplantation of nerves, sciatic scoliosis and Achilles bursitis.

HAROLD BENNETT(1837-1907)

Studied at Trinity College, Dublin. He collected specimens of bone pathology and with these wrote a paper on fractures of the metacarpal bones in 1882. In this paper Bennett described his eponymous fracture dislocation of the base of the thumb metacarpal. Bennett is said to have introduced antisepsis to Dublin and to have performed many osteotomies for rickets. He became President of the Royal College of Surgeons of Ireland.

HUGH THOMAS (1834-1891)

If you could only read about one person in the history of Orthopaedics, then you would have to read about Hugh Owen Thomas, the father of British Orthopaedics surgical instruments. He believed in enforced, prolonged and uninterrupted rest for the treatment of tuberculous joints. Thomas developed a great number of splints in order to achieve this. These include, the cervical collar, metatarsal bar, heel wedge and knee splint. Many of these are still in use, such as the Thomas splint. Thomas was also able to recognise early cases of hip disease. He was the first to demonstrate concealed flexion of the hip joint and a way of unmasking this by performing the Thomas Test.

It should be remembered that Hugh Owen Thomas had studied Medicine and was interested in litholopaxy and the management of acute abdomen as well as Orthopaedics. It has been said that the medical profession might not have practised the black art of bone setting, if Hugh Owen Thomas had not graduated from a Medical school.

SIR WILLIAM ARBUTHNOT LANE (1856-1938)

Lane was a Scot from Inverness who trained and later worked at Guy's Hospital in London. Lane is known for his attempts at improving alignment of fractures by using internal fixation. He started off using silver wire, then he used steel screws and this was followed by the use of plates and screws. Lane was said to have been eccentric, regarding humans as machines and performed total colectomies as a cure for auto-intoxication. He also initiated the programmes of health education that are present today. Lane wrote columns in the newspapers, held public lectures and improved the distribution of fruit and vegetables.

FRITZ DE QUERVAIN (1868-1940

De Quervain was born at Sion in the Valais Canton of Switzerland. He studied at Berne and succeeded Kocher as Professor of Surgery there. In 1895, De Quervain described a form of chronic tenovaginitis, which is now known as De Quervain's stenosing tenovaginitis. Like Kocher, he studied thyroid disease and is responsible for the introduction of iodised table salt.

FRIEDRICH TRENDELENBURG (1844-1924)

Trendelenburg was born in Berlin. He studied Medicine in Glasgow and in Berlin. Trendelenburg's name is associated with the Trendelenburg sign and the Trendelenburg gait, which he described in association with coxo-femoral incompetence in 1895. Trendelenburg also devised pulmonary embolectomy, but it was one of his pupils, Kirshner who first met success with the procedure many years later in 1924.

PIERRE MARIE (1853-1940)

Marie was born in Paris, he worked for Charcot and eventually succeeded him as Professor of Neurology at Salpetriere. Marie described peroneal muscular a trophy (Charot-Marie-Tooth disease). He was the first to associate acromegaly with a pituitary tumour in 1886. In 1980, he described hypertrophic pulmonary osteoarthropathy. In 1898, he gave the first account of cranio-cleidal dystosis and noted the partial aplasia of the clavicles, the increased skull diameter, the disordered dentition and the failure of ossification at the fontanelles. Also in 1898, he published a classic paper on ankylosing spondylitis, which he refereed to as spondylosis rhizomelique. Marie believed that poliomyelitis was infectious.

LOUIS XAVIER EDOUARD LEOPOLD OLLIER (1830-1900)

Ollier was born in Vans in Ardeche and studied at Lyons and ontpellier. Ollier, like Macewen, performed pioneering bone grafts. Although both were successful, their methods and the theory behind them were in fierce opposition. In 1877, Ollier suggested that bone growth may be inhibited in order to correct certain deformities by resecting the epiphyeal plate. In 1899, Ollier first described dyschondroplasia or Ollier's Disease. Ollier researched bone growth to an enormous extent and believed that it might be possible one day to treat patients by stimulating their cartilage to ossify.

WILHELM CONRAD RONTGEN (1845-1923)

Although Rontgen was a professor of Physics at

Wurzburg, his discovery of X-rays (Rontgen rays) and their use has provided an enormous contribution to Orthopaedics and is still of great value to Orthopaedic practice. The first radiography that Rontgen took was of his wife's hand on the 22nd of December 1895. This was allegedly her Christmas present. Rontgen received the Nobel Prize for his discovery

MODERN ERA (2OTH CENTURY ORTHOPAEDICS) THE TURN OF THE CENTURY.

The early 1900's can be seen as a great turning point for Orthopaedics. The discovery of the X-ray almost marked 1900 and Orthopaedics itself was only now being seen as a true specialty of its own. The British still dominated Orthopaedic developments, but the new world had now reached maturity and there were increasingly more contributions being made by the Americans. The bloom of understanding, with the introduction of the X-ray, was not as dramatic as expected. Instead, the turn of the century was marked by new institutions and associations that sought to mark Orthopaedic Surgery as an individual and growing specialty.

One area where there was an evident surge of new information with the introduction of the X-ray was that of osteochondritis and osteonecrosis. Although the German George Clemens Perthes took the first X-rays of Perthes' disease in 1898, Perthes' assistant did not publish these until 1914. In 1903, Robert Osgood (1873-1956) of Boston first described a lesion of the tibial tubercle occurring during adolescence. This is now known as Osgood-Schlatter's disease. The German, Albert Hoffa (1859-1907), stressed in his book of 1902, that excision of the femoral head for cases of hip disease should not be routine. He had realised that not all cases of hip disease were tuberculous. Hoffa was interested in scoliosis, but has his name associated with the hypertrophy of the infrapatellar fat pad that is now called Hoffa's disease. Another German by the name of Georg Axhausen (1877-1960), is noted as the first to use the word aseptic necrosis. It must be stressed that necrotic bone was frequent at this time, for there were no antibiotics, and any study into a non-infectious bone necrosis was innovative. In an article he published in 1910,Axhausen wrote that necrosis occurred at the bone-ends of every fracture, and that this stimulated and was replaced by periosteal proliferation. He also believed that focal necrosis of subchondral bone caused changes in the overlying articular cartilage which lead to arthritis deformans. It was not until the 1950's that Axhausen's term of aseptic necrosis was replaced by the term avascular necrosis.

Jacques Calve (1975-1954) of Berck, France, Arthur T. Legg (1874-1939) of Boston, U.S.A. and *George Perthes* of Tubingen, Germany, are all said to have described Perthes' disease in 1910. Hence this disease is sometimes referred to as Calve-Legg-Perthes disease. With the help of radiography, Calve realised that some cases of tuberculous hips in children were actually cases of coxa plana. He saw that these rare cases of hip irritability had X-ray evidence of coxa vara, hypertrophy of the femoral head, increased density, fragmentation and flattening of the epiphysis. He noted that the disease was of short clinical duration, had good recovery, did not relapse and was not associated with adenopathy or abscess. Calve also described vertebral osteochondritis with collapse, which he attributed to vascular changes subsequent to trauma (although we now know that eosinophil granuloma is the common cause). Legg had wide interests, but is best known for the eight papers that he published on coxa plana. He included a treatment of traction hip splint and noted the limited degrees of movement.

The early 1900's was also a time of improvement for spinal surgery. Russell A. Hibbs (1869-1932), was from the New York Orthopaedic Hospital and in 1911, published a report on a technique of spinal fusion that he had developed. Hibbs performed the first spinal fusion for tuberculosis and later performed a similar procedure for scoliosis. Joel Goldthwait (1867-1961) was another of the great Boston orthopaedists. He had a major interest in posture and in 1911, published a laminectomy from L1 to S3 performed on a man who developed bilateral sciatica followed by paraplegia after a lifting strain.

Probably the most important figure at the turn of the century was Sir Robert Jones (1855-1933). Indeed many would argue that he was the greatest orthopaedic surgeon that the world had ever seen. It was said that when Jones operated, Time stood still. Jones was a nephew of the great Hugh Owen Thomas and became one of his apprentices in Liverpool. In 1896, Jones published the first report of the clinical use of an X-ray to locate a bullet in a wrist. He founded several associations and Orthopaedic hospitals. Jones wrote several important books such as Injuries of Joints in 1915 and Notes on Military Orthopaedics in 1917. His textbook Orthopaedic Surgery is said to be the first to have dealt systematically with the diagnosis and treatment of fresh fractures. In World War I, Jones headed the orthopaedic section of the British Forces. Jones was an advocate of tendon transplantation, bone grafting, and other conservative, restorative procedures.

WORLD WAR ONE.

It must be noted that war has played an important part in Orthopaedic history. Many of our greatest contributors were military surgeons and it is remarkable how much we have learnt about ancient Greek orthopaedics through Homer's description of the Trojan War alone. It is interesting to note that many of the achievements during and after World War I were not related directly to traumatic injuries received at war. However, it can be said that Orthopaedics was definitely seen as a separate specialty after World War I and that this was the first major war where aseptic techniques were saving many more lives than in the past wars.

Jules Tinel (1879-1952), of Rouen and Paris, was a neurologist in the First World War and first described Tinel's sign in 1917. This related to nerve injuries, and on percussion of the nerve below the site of nerve injury, formication was elicited if nerve fibres were degenerating. If this was absent, there was a bad prognosis. Paul Budd Magnuson (1884-1968) was an American who assisted in orthopaedic services in World War I and with this and his own practice in the slaughterhouse area acquired much experience in trauma. He wrote a textbook called Fractures and in this described complete debridement of the knee joint for osteoarthrosis, including synovitis and other degenerative lesions. This involved the removal of osteophytes and damaged menisci, shaving of the cartilage to the bare bone and if necessary, narrowing of the patella. Following the war, an American by the name of Paul N. Jepson (1893-1949) continued Volkmann's fine work and reproduced ischaemic contracture in animals. In his paper of 1921, he wrote that ischaemic contractures could be prevented by prompt surgical decompression. In 1927, a Russian called M. J. Arinkin introduced sternal puncture as a diagnostic procedure while working at the Military Medical Clinic in Leningrad. This can be applied in the study of metastatic bone disease.In the chain of great surgeons that followed Hugh Owen Thomas.

The immediate challenges facing the modern orthopaedic surgeon are : The Human genome Project (sequencing the 23 human chromosomes; due for completion 2003) with the promise of gene therapy, human cloning and bovine prosthetic implants; corporate control of the practice of medicine with surgeons answering to corporate bosses rather than their patients and colleagues; the rise of the Internet with a more efficient interchange of information and the Global Burden of Disease where motor vehicle accidents are going to be the third greatest burden of morbidity by the year 2020. These challenges are there and are somewhat daunting but certainly less frightening than many that faced our proud forebears and teachers. If alive, John Hunter would be in the forefront of these scientific developments and I suspect, chide us for our tardiness in embracing such challenges. In some ways, Orthopaedics has lost its way by producing surgical technicians (surgeons merely content to operate) with not enough commitment to fundamental biomedical research and the protection of their craft.

"ANATOMY, STRUCTURE AND FUNCTION OF A CHANNEL AND CYLINDER" (THE MEATUS AND THE TUBE)

Abdul Ahad M.S.; Shameema Jabeen M.D.; Masarat Rasool M.B.B.S.

A tube means a long hollow cylinder for holding or conveyance of fluid. The word is used in Anatomy to name the auditory tube (Pharyngotympanic or Eustachian tube) which conveyes air to equalise the presure of air between the middle ear cavity and the pharynx and drains the mucous from the mastoid air cells and the tympanic cavity.

A meatus means a channel / passage which is used as a word / name to recognise the external auditory meatus which conveys normally sounds (if not the backbite / polluted air / insects also.

The differences between the two on Anatominal basis are penned down hereunder:-

External Auditory Meatus (EAM)	Auditory Tube (A.T.)
1. The external auditory meatus stops at the tympanic membrane and looks like a tall narrow goblet.	It is a tube open at both the ends though the walls at the medial end remain opposite each other except during deglutition.
2. The EAM develops from the dorsal part of the first ectodermal cleft and is very short at birth. Its deepest part is represented by the auditory tubercles-a collection of cells which later canalise to form the bony part of the canal. EAM is fully developed by the age of 9 years.	It develops from the medial part of tubo-tympanic recess which elongates and communicates with developing pharynx at the tubal elevation. At the lateral end it opens into the anterior wall of the tympnum via the bony part.
3. External auditory meatus is a tunnel in the temporal bone that extends from the concha of the auricle to the tympanic membrane medially. It is S- shaped and looks like a tall, narrow and twisted goblet. It is directed medially, forwards and slightly upwards. It is about 2.5 cms long. The lateral part is 8 mm long and cartilagenous whereas the medial bony part is 16 mm long. The meatus has two constrictions. The Isthmus is in the osseous part 2 Cms from the bottom of the concha whereas the second constriction is near the medial end of the cartilagenous part. The osseous part on the whole is narrower than the bony part. It is directed anteromedially, slightly downwards with the posterosuperior convexity. The medial end of this osseous part is narrower. The lateral end is most rough to give attachment to meatal cartilage by ligaments. The meatus is lined with skin. In the subcutaneous part (which is thick) are modified glands, the ceruminous glands to prevent (a) Maceration by retaining water and (b) entry of insects.	It passes downwards forwards and medially (30° to the horizontal and 45° to the sagittal plane). It is 36 mm long. The part near the ear is 12 mm (1/3rd) and the part towards the pharynx is 24 mm in length (2/3rd). The bony part narrows as it passes medially from the tympanic cavity and has a (Narrowing) isthmus at its junction with the cartilagenous part. This end of the bone is jagged to give attachment to the cartilagenous part--the two parts meeting at an obtuse angle. In new borne it is half in size, horizontal with the bony part, shorter but much wider. There is no tubal elevation. The tube is lined by ciliated columnar epithelium which is thin in the osseous part and thick in the cartilagenous part due to the presence of mucous glands. The pharyngeal orifice is associated with tubal tonsil of variable thickness.
4. The osseous part is formed by C-shaped tympnic part of the temporal bone. Only postero-superior part is formed by the squamous and petrous parts of the temporal bone.	Pertrous temporal bone is canalised to form the osseous part.
5. The outer cartilagenous part (Meatal cartilage) is deficient posterosuperiorly and the gap is occupied by	The cartilagenous part is formed by a triangular plate of cartilage which is fixed to the base of skull in the groove

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collagen. Anterior part has 2-3 deep fissures.	between the greater wing of sphenoid and posterior part of temporal bone. Inferolaterally the tube is completed by fibrous membrane. This part widens towards pharynx. The opening is pharyngeal 1-1.25 cm below and behind the posterior end of the inferior nasal concha. This orifice is directed downwards and backwards.
6. The cartilaginous part does not have any muscular attachment.	Three muscles take origin from the cartilagenous part of the tube, I. Tensor veli palatini. II. Levator veli palatine and III. Salpingopharyngeus.
7. **NERVE SUPPLY** i. Anterior and superior walls are supplied by auriculotemporal nerve (branch of mandibular nerve. ii. Posteroinferior part is supplied by auricular branch of Vagus (Arnold's Nerve). ii. Facial nerve may also supply via communication with vagus (It explains herpes zoster in the meatus due to Facial Ganglion involvement. Tympanic membrane is also supplied by these nerves plus glossopharyngeal).	i. Pharyngeal branch of Maxillary nerve via pterygoplatine ganglion. ii. Tympanic plexus (Lesser petrosal of 9th nerve plus Sympathetics) (Needs further study to confirm other source like facial......)
8. **ARTERIAL SUPPLY** i. Posterior auricular Artery. ii. Syperficial temporal iii. Deep auricular (Branch of Maxillary Artery)	i. Ascending Pharyngeal branch of Internal carotid. ii. Middle meningeal. iii. Artery of the pterygoid canal.
9. **VENOUS DRAINAGE:** i. External jagular vein. ii. Maxillary vein. iii. Pterygoid plexus of veins.	i. Pterygoid plexus of veins.
10. **LYMPHATIC DRAINAGE:** The following lymphnodes serve as the satellite nodes. i. Parotid ii. Retroauricular. iii. Superficial cervical.	The median and the two lateral retropharyngeal nodes are the satellite nodes for this tube (nasopharynx and atlantoaxial and atlanto-occipital joints are drained by these nodes).
11. **IMPORTANT RELATIONS.** Three important bony relations:- i. Anteriorly head of the mandible seperated by a small part of parotid gland. ii. Middle cranial fossa above. iii. Mastoid air cells are posterior to the bony part and the mastoid antrum is seperated from it anterio-inferiorly by a thin lamina of bone (1-2mm) which is the only osseous resistance for transmeatal approach for aural surgery.	Three muscular: i. Anterolaterally is tensor veli palatini. ii. Posteromedially is lavator veli palatini. iii. Inferiorly is salpingopharyngeus. Tensor veli palatini separates the PTT from OTIC ganglion, mandibular nerve and its branches, chorda tympani and middle meningeal artery.
12. **FUNCTIONS:** ***External Auditory Meatus is*** Filled with air which is warmer than the air outside. It could have some role in addition to:- i. S-shaped EAM protects the tympanic membrane. ii. Intensity of sound is increased by about 10 Decibels. iii. Ceru-minous glands EAM-Secretecerumen which discourages entry of insects and protects skin of EAM by imbibing water. The entry could be prevented by the isthmus and other narrowing for the bigger insects. iv. Commonest function held is conducton of sound to the drum of the ear. v. The hair present in the EAM prevents entry of dust, insects and its excessive growth in some could (Yet to be confirmed) effect transmission of sound.	i. Pharyngotympanic tube opens during deglutition and helps to balance the pressure of the air between the pharynx and the middle ear cavity. ii. The tube helps to drain the secretions from the middle ear towards the pharynx.

HYDATID DISEASE: CURRENT STATUS AND RECENT ADVANCES

Mohammad Sultan Khuroo, MD, FRCP (Edin), MACP.

Hydatid disease, also known as echinococcosis or hydatidosis, is caused by infection with larva (metacestode) of the tapeworms of the genus Echinococcus. Within the genus Echinococcus, four species are recognized: E. granulosus which causes cystic hydatid disease; E. multilocularis which causes alveolar hydatid disease; E. vogeli and E. oligarthrus both of which cause polycystic hydatid disease (Table 1).[1,2]

Hydatid disease is a public health problem in Asia, the Mediterranean, South America and Africa. With mmigration, the prevalence of the disease has increased in Europe and North America in recent years.[3-8] There has been considerable recent progress in the diagnosis and surgical treatment of hydatid disease.[2,9,10] Drug therapy and percutaneous aspiration have been successfully used to treat hydatid disease, and long-term results have been encouraging.[11,12] In view of the above, clinicians need to be aware of the prevelance of hydatid disease and the recent advances in its treatment.

Life Cycle

Echinococcus species require two hosts for completion of the life cycle. The definitive hosts are carnivores harboring mature tapeworms in the intestines. Metacestodes develop in the liver and other viscera of intermedate hosts, and under natural conditions, host assemblages are linked together in predator-prey relationship. Human beings become incidental intermediate hosts and do not complete the life cycle of the parasite.

Adult Echinococcus species are small tapeworms measuring 2-11 mm in length. Each adult worm consists of a protoscolex, the cephalic end for attachment and 2 to 5 segments, the proglottids. The scolex bears two rows of keratinized hooks and fore suckers. The proglottids are produced continuously from the neck region immediately behind the scolex. The terminal gravid proglottid contains several hundred fertilized eggs and is shed every 7 to 14 days. Ovoid eggs have a diameter of 30-36 um and contain fully differentiated oncosphere. Eggs are shed with the feces of the infected definitive host. When ingested by a suitable intermediate host, the oncospheres hatch in the small intestines, become activated, penetrate the epithelial layer and migrate via blood or lymphatic vessels to the viscera. In the viscera, the oncosphere undergoes a series of reorganization events involving degeneration, cell proliferation, differentiation and vesiculation, resulting in the formation of primary vesicle (metacestode). Metacestode develop into hydatid cyst by a process unique for each species of Echinococcus. Scoleces are formed within the cysts which when ingested by the definitive host (eating offal of intermediate host) develop into adult tapeworms.

Cystic Hydatid Disease

Geographic Distribution (Figure 1)

E. Granulosus is a cosmopoltan parasite and has at least six genetcally distinct strains, two of which are irelevant to human infection. The sylvan strain is maintained in wolves and wild ungulates in Northern Alaska, Canada, Scandinavia and Eurasia. The pastoral strain is maintained in dogs and domestic ungulates throughout the world. Most of the human infections occur from pastrol strain maintained between dog and sheep. Endemic regions of human hydatid dsease include the southern parts of South America (Uruguay and Argentina), the Mediterranean region, the Middle East, many parts of Africa (especially Kenya), Southern and Central Russia, Central Asia and many regions in China (especially Xinjinag province). The majority of patients reported in Austria, Germany and Switzerland are immigrants from the Mediterranean region. The disease has been eradicated in Iceland and disease load Cyprus by control measures. In the United States, most infections are seen in immigrants from enedmic areas, but autochthonous transmission is currently recognized in Alaska, California, Utah, Arizona and New Mexico. The annual iincdence of human infecton in endemic areas varies widely (<1 to 220 per 100,000 per annum) from region to region.[1-8,13]

Pathogenesis and Pathology

Dogs nfected with the tapeworm, E. granulosus, pass eggs in their feces and the cestode eggs adhere to the hair, muzzle and paws of the animal. Humans become infected in the course of playful and/or intimate contact with the infected animal. The eggs hatch in the small intestine, liberate oncosphere that burrow the mucosa, migrate via vessels or lymphatics to distant sites, and develop into a metacestode. The metacestode develops into a unilocular

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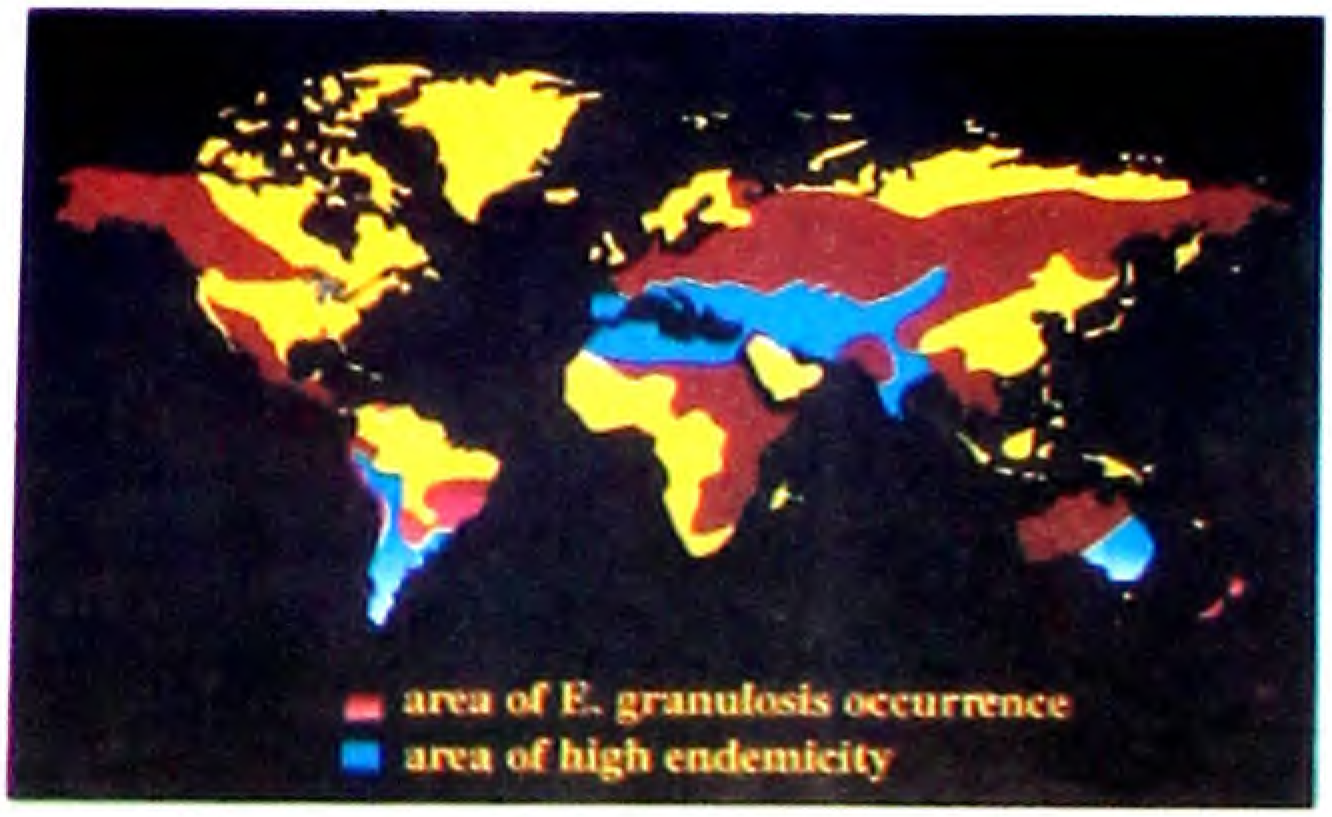

FIGURE 1. Geographical distribution of cystic hydatid disease.

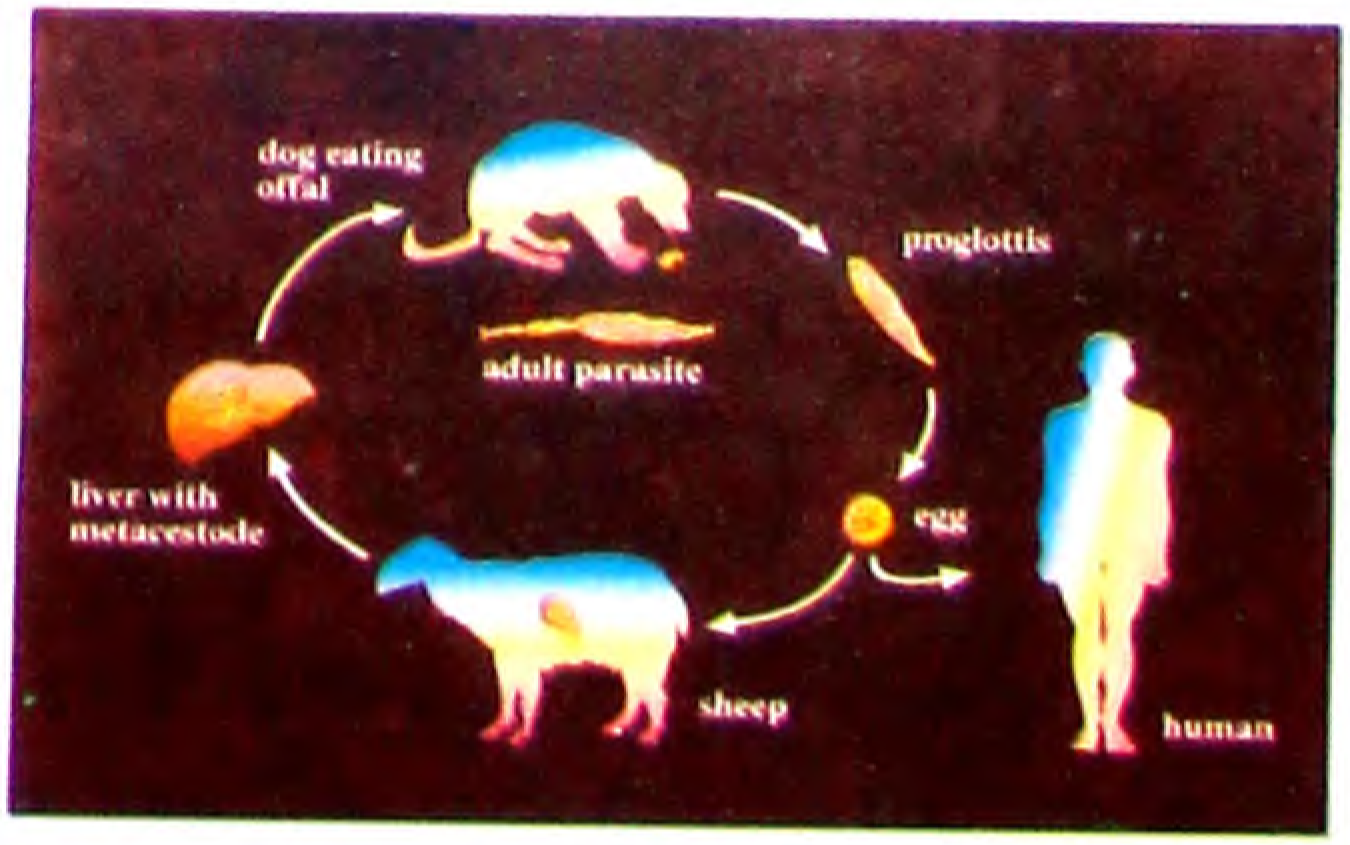

FIGURE 2. Life cycle of *Echinococcus granulosus*. The parasite completes the life cycle between dog, the definitive host and sheep, the intermediate host. Human beings become incidental intermediate hosts and do not complete the life cycle of the parasite.

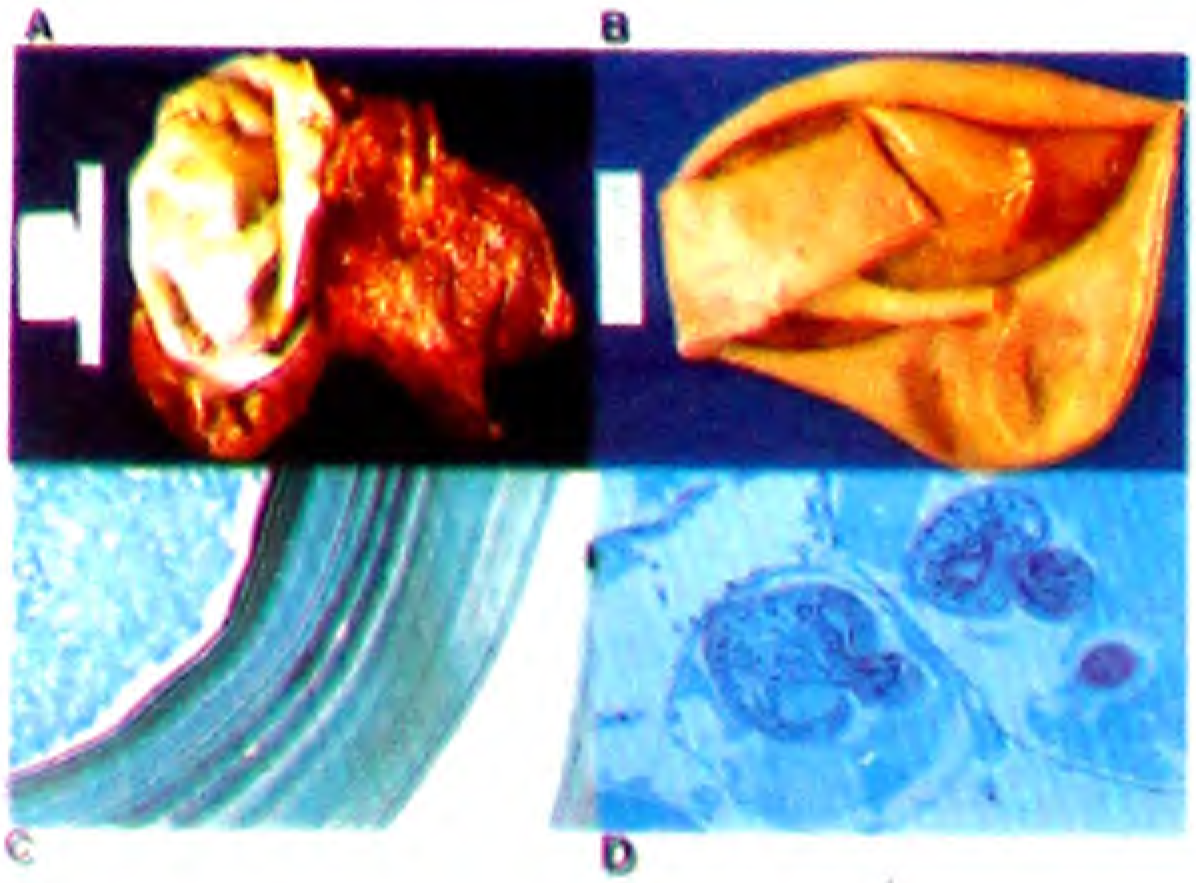

FIGURE 3. Morphology of cystic hydatid disease. Panel A shows resected left upper lobe lung with a large hydatid cyst. Panel B shows detached ivory-white laminated membrane of the cyst. Panel C shows histological section of the laminated membrane. The inner dark staining layer is the germinal layer of the cyst. Panel D shows two evaginated protoscolices within brood capsules.

fluid-filled cyst (Figure 2).[2,10,13]

E. granulosus cysts following primary infecton may inhabit any anatomic site. The two most common organs involved are liver (65%) and lungs (25%). Cysts of the sylvan strain predomnantliiy localize in the lungs. Other less common sites affected by cysts include the muscles (5%), spleen (1%), bones (3%), kidneys (2%), heart (1%), pancreas (1%) and central nervous system (1%). The majority of patients have single-organ nvolvement (87%) and harbor a solitary cyst (72%). Secondary infection follows cyst rupture and scolices can grow in the peritoneum, pleura, broncial tree and bile ducts or be carried via blood stream to distant organs. Cyst size ranges from 1 to 15 cm and cyst growth varies from 1 to 30 mm per year.[13,14]

Hydatid cyst wall consists of inner germinal layer and outer laminated membrane (Figure 3). Surrounding the wall is the pericyst, which has an outer zone of compressed liver cells, middle zone of heavy lymphoplasmocytic infiltrate and inner tough zone formed of collagen. The cyst fluid is crystal clear with a specific gravity of 1012 and contains salts, enzymes, proteins, and toxic substances, and is antigenic. The germinal layer forms small cellular masses projecting into the cyst cavity, which vesiculate to form brood capsules. In each brood capsule, multiple protoscolices (head of the future worm) develop on the inner surface of their walls by asexual budding. Brood capsules and protoscolices may be released into the cyst fluid to form hydatid sand. Protoscolices have a potential to develop into adult tapeworm within the gut of the definitive host, differentiate into secondary cysts when released following cyst rupture (ectogenic vesiculation), and form daughter cysts within the mother cyst by endogenic vesiculation. each daughter cyst is a true replica of the mother cyst and has a germinal layer and laminated membrane and contains brood capsules and protoscolices. Daughter cysts may be packed together in the mother cyst (multivesicular cyst) or float freely in the mother cyst cavity. In older cysts, the contents degenerate forming a gelatinous amber-colored structure called the matrix. The matrix gives a pseudo-tumor appearance on imaging tools and can be confused with pus by the surgeon. Calcification can occur in the pericyst, mother cyst and daughter cyst. Calcification of endocyst indicates that the cyst is nonviable. However, calcification of the pericyst is found in one-third of cysts and occurs in the stages of cyst development.[2]

Clinical Manifestations

Infections are usually acquired during childhood, however, most cases of cystic hydatid disease present in adults in the third to fifth decades of life, with equal sex distribution. The clinical features depend upon the organ involved, site of organ involvement, stage of cyst

TABLE 1 *Human hydatid disease.*

	Cystic	Alveolar	Polycystic	
Agent				
Species	*Echinococcus granulosus*	*Echinococcus multiocularis*	*Echinococcus vogeli*	*Echinococcus oligarthra*
Adult size	2-7 mm	4-11 mm	1.9-3.7 mm	1.9-2.9 mm
Definitive host	Dog, wolf, jackal, lion	Foxes	Bush dogs	Wild felid
Intermediate host	Sheep, cattle	Rodents	Paca	Spiny rats
Human disease				
Occurrence	Common	Uncommon	Rare	Extremely rare
Mode of infection	Contact with infected dogs	Contact with fur from infected fox, ingestion of contaminated wild berries	Contact with hunting dogs fed on paca	Contact with infected cats
Lesion appearance	Fluid filled unilocular cyst	Solid mass	Polycystic fluid filled	Polycystic fluid filled
Calcification	Ring type (30%)	Microcalcification or plaque like foci (70%)	Annular, bizarre, amorphous (50%)	Not known
Organs involved				
Primary	Liver (65%), lung (25%), others	Liver (100%)	Liver (100%)	Extrahepatic (orbit others)
Spread	By rupture	Contiguous, metastatic to lung, brain	Contiguous	Contiguous
Presentation	No symptoms, mass effect, rupture, infection	Invasive liver mass, liver failure, cholestasis, Budd chiari, portal hypertension, lung and brain involvement	Liver masses, liver failure, cholestasis, Budd chiari syndrome, portal hypertension	Exophthalmos, soft tissue mass
Mortality untreated (10 years)	Unusual (<5%) (anaphylaxis/complicated cyst)	High (75-100%)	High (>75%)	Not known

development and viability of the cyst contents. The majority of the cysts produce no symptoms and are detected as incidental findings on routine imaging or at autopsy. Symptoms of the disease are related to expanding mass, pressure on adjacent structures, infection and rupture of cyst contents into surrounding body cavities.[15]

Uncomplicated liver cysts usually present with dull ache in the right upper quadrant or a feeling of abdominal swelling or distension. Clinical examination is unremarkable except for enlarged liver with a rounded mass felt on its surface. A hydatid thrill may be elicited on a large mass pointing to its fluid content. Rarely, the mass is clacified giving it a hard consistency, and needs to be differentiated from a primary or secondary liver tumor.[13,15] A large cyst in the hilar region can compress the common hepatic duct causing cholestasis.[16] Hepatic vein or inferior vena cava compression can cause Budd-Chiari syndrome.[17] Cyst rupture into the biliary tree causes obstruction of the ducts by daughter cysts and laminated membranes and presents as biliary colic, cholangitis and progressive cholestasis.[18] Cyst contents may block the ampullary orifice causing acute pancreatitis. Cyst rupture into the peritoneum causes immediate life-threatening anaphylaxis and if the person survivers, the scolecess implant on the peritoneal surface resulting in extensive peritoneal hydatidosis.[19,20] A cyst can rupture into the pleural cavity or lung and can establish cystobroncial fistula.[15] Rarely, cyst can grow on to the skin along a sinus tract following surgery or percutaneous drainage of the cyst.[21] Infection of the cyst follows tear in the laminated membrane and soilage of the cyst with bile or blood. Infected hydatid cyst has clinical features of a liver abscess.[15]

Intact hydatid cysts in the lungs often cause no symptoms. However, a large cyst may cause pressure symptoms and lead to cough and hemoptysis. Complications arise when the cyst ruptures either into the bronchial tree or pleura. Bronchial rupture manifests with cough, chest pain and dyspnea. Spontaneous cures are known, as laminated membranes may be coughed up leading to collapse and disappearance of the cyst cavity. Rupture of cyst contents into the pleural cavity may cause allergic reactions including anaphylaxis, pleural effusion, pleural implanation and empyema. Infection of the cyst follows leakage of the cyst and presents as lung abscess.[13,15]

Involvement of other organs can lead to varied manifestations, namely soft tissue fluctuant swelling (muscle cysts), raised intracranial pressure and focal epilepsy (cerebral cysts), segmental portal hypertension (splenic cysts), acute pancreatitis and obstructive jaundice (pancreatic cysts), bone pain, swelling and pathological fractures (bone cysts), paraplegia (vertebral body cysts), conduction defects, pericardial effusion and cardiac tymponade (cardiac cysts), loin pain and hematuria (kidney cysts), unilateral exophthalmos and blindness (orbital cyst).[22-24]

Diagnosis

Routine hematological and biochemical tests do not help in diagnosis of cystic hydatid disease. Serology and imaging tools establish diagnosis.

Serology

Serology detects specific serum antibodies or circulating antigen by a variety of immunodiagnostic methods, of these, the enzyme immunosorbent assay (ELISA) employing hydatid fluid antigen for detection of echinococcal antibodies (IgG) in the serum is the most widely used. This test has a sensitivity of 80%-100% and specificity of 88%-96% in hepatic cysts. However, the sensitivity of this test is around 50%-56% in pulmonary cysts and 25%-56% in cysts involving other organs. False-positive test can occur in normal persons from endemic areas and in those with other parasitic infections.[25-26] Positive test results need confirmation by the arc-5 immunoelectrophoretic (IEP) test, which detects antibodies against immunodominant and specific antigen (antigen 5) of the cestode. This test does not cross-react with noncestode parasites. Cross-reaction with Taenia solium cysticercosis does occur and need to be differentiated in regions where both infections are prevalent.[27] Serological tests have limitations in monitoring patients after surgery, percutaneous drainage in monitoring patients after siurgery, percutaneous drainage or drug therapy. The antibody titers rise following surgery and percutaneous drainage. The titers start falling at 3 months and become negative in a period of 12-24 months.[12]

PCR using recombinant DNA antigens is valuable in defining particular species of E. granulosus. This has been effectively applied in the control of the disease and development of an animal vaccine.[28]

Imaging

A number of imaging tools are available to define hydatid cysts and need to be utilized judiciously. The selection of these tools depend upon the organ to be imaged, the status of the cyst (intact, ruptured or infected) and adjacent structures which need to be assessed.[2,18,29] Plain radiography has a role in lung and bone cysts. A plain film of chest shows intact cyst as sharply dermarcated round-to-oval homogenous mass of variable size. Pulmonary cysts do not calcify, and daughter cyst formation is rare. Rupture of the cyst wall may present as the air meniscus sign (radiolucent air shadow between pericyst and intact endocyst); the double arch sign (air delineating wall of the pericyst, the outer arch and detached wall of the endocyst, the inner arch); and the water lily sign (collapsed wavy endocyst membranes floating on the top of remaining cyst fluid). Bone cysts occur in the pelvis, vertebral column, long bones and skull, and cause expansion and destruction of the bone without the bone. The lesion often crosses the joints and involves adjacent bones. A plain film of abdomen may show liver cyst calcification. This occurs in the ectocyst and is typically curvilinear (egg shell calcification). Encocyst calcification is spotty in nature and appears as speckled calcified densities. Cysts at other sites may calcify and are shown on plain films of the organs.[30-31]

Abdominal cysts, particularly in the liver and cysts in soft tissues, are well visualized by ultrasound. Cysts may appear univesicular, rounded with well-defined margins containing pure fluid, or multivesicular (daugher cysts) with pure fluid collection iin each vescle. Cysts may have hyperechoic solid pattern (pseudotumor appearance) or may have reflective wall suggestive of calcifiction. rupture of liver cysts into the bile ducts can be suspected on ultrasound. Ultrasound appearances include dilated bile ducts, non-shadowing echogenic structures within the ducts and loss of continuity of the cyst wall adjacent to the bile duct representing site of communication. However, ERCP is the recommended tool and reveals filling defects of varying shape and size in the dilated bile ducts and leakage of contrast medium into the cyst cavity.[26]

Computed tomography is the method of choice for detecting lung, brain and bone cysts. It has an advantage over ultrasound for cysts at other sites for better documentation of site, size and structure of cyst and for monitoring of lesions during chemotherapy and following surgery, daughter cysts and detachment of membranes, if any. Magnetic resonance imaging may show a characteristic intense rim. Angiography and scientiigraphy have no major advantage over above imaging tools and are rarely employed in this setting.[2-4]

Diagnostic puncture of cysts is only justified when imaging and serological tests do not permit discrimination between hydatid cyst and neoplasm. It carries risk of anaphylaxis and spillage of cyst contents with secondary echinococcosis.[31-32]

Treatment

Treatment options for cystici hydatid disease are surgery, drug therapy and percutaneous drainage. Surgery has the potential to remove cysts and lead to complete cure. Surgical procedures of choice include cystectomy with removal of the germinal and laminated layers and preservation of pericyst. Cavity is either tightly drained, closed or packed with omentum (omentoplasty). Occasionally, cyst is removed by total pericystectomy, partial hepatectomy or segmentectomy. Surgery is the treatment of choice n large hepatic cysts with multiple daughter cysts, subcapsular or pedenculated liver cysts, infected liver cysts, cysts communicating with bile ducts, and cysts in the lungs, brain, kidney, bones and other organs. Surgery is contraindicated in patients at extremes of age, pregnant women, patients with co-morbid diseases and in those with multiple cysts and cysts, which are difficult to access. Operative mortality varies from 0.5%-4.% in centers with adequate medical and surgical facilities. Cyst fluid spillage can occur during surgery resulting in anaphylaxis and/or secondary echinococcosis (2%-25% of cases).[10,33-35]

Two benzimidazoles (mebendzole and albendzole) and praziquantel, and isoquinolone derivative, have scolicidal activity and have been used in over 1000 well-documented patients with cystic hydatid disease. Mebendazole is poorly absorbed, and soclicidal drug levels are achieved with a daily dose of 4-50 mg/kg adminsitered orally in divided doses. Albendazole is better absorbed, and the usual daily dose is 10-15 mg per kg. Drug dosing with a fat-rich meal improves intestinal absorption. Cyclic treatment (three 1-month courses with intervals of 14 days) has been widely used. Recent data show that uninterrupted drug therapy for 3-6 months has better efficacy with no increase in adverse effects. Praziquantel is administered at a dose of 40 mg/kg once a week concomitantly with benzimidazoles, and has been shown to increase the soclicidal activity of the imidazoles. Chemotherapy achieves a cyst disappearance of 30%, partial response in another 20% and no response in 40%. It is effectve in small cysts (<4 cm diameter), cysts with thin walls and in younger patients. It is indicated n patients who are high risks for surgery, in patients with multiple peritoneal cysts, to prevent secondary echinococcosis after spillage during surgery, and as a concomitant therapy with ipercutaneous drainage. Imidazoles are hepatotoxic, can cause neutropenia, thrombocytopenia, alopecia and are potentially embryotoxic and teratogenic.[11,36-41]

Recently, percutaneous drainage of hepatic hydatid cysts, popularly known in Europe as the PAIR (Puncture, Aspiration, Installation of scolicidal agent and Reaspiration) technique has gained acceptance.[42-48] The procedure is minimally invasive, cost-effective, involves reduced hospital stay and and has less morbidity and mortality than surgery.[12] It is the treatment of choice in patients with hepatic hydatid cysts who either refuse surgery or have significant co-morbid diseases.[36] The procedure is highly effective in intact univesicular and multivesicular cysts with few large daughter cysts, cysts with diameters of over 5 cm and multiple cysts in different liver segments. Cysts relapsing after surgery or failed to regress following chemotherapy are amenable to percutaneous drainage. The procedure cannot be done in superficial or pedenculated cysts, small volume cysts, cysts with honeycomb appearance (multiple small daughter cysts), cysts with dominant solid component, and those with infection or communicating with bile ducts. The procedure is associated with possible complications of liver puncture (bleeding, bile peritonitis), anaphylaxis, allergic reactions and biliary communciations.[36] Inadvertent installation of sclerosing agent into a cyst with biliary communiation can cause sclerosing cholangitis.[49,50] Long-term follow up has shown that secondary echinococcosis does not occur following percutaneous drainage.[21]

Endoscopic therapy has been used in a small number of patients with hepatic cysts which have ruptured into bile ducts. At ERCP, encoscopic sphincterotomy is performed; laminated membranes and daughter cysts are extracted, and a nasobiliary drain is placed in the bile duct. The cyst regresses and disappears over the next 4-6 weeks.[12,20,44,51] Laparoscopic removal of hepatic cysts have been reported and liver transplant has been performed in extensive hepatc hydatidosis not amenable to surgical resecton. None of these modalites have been evaluated in controlled trials.[52,53]

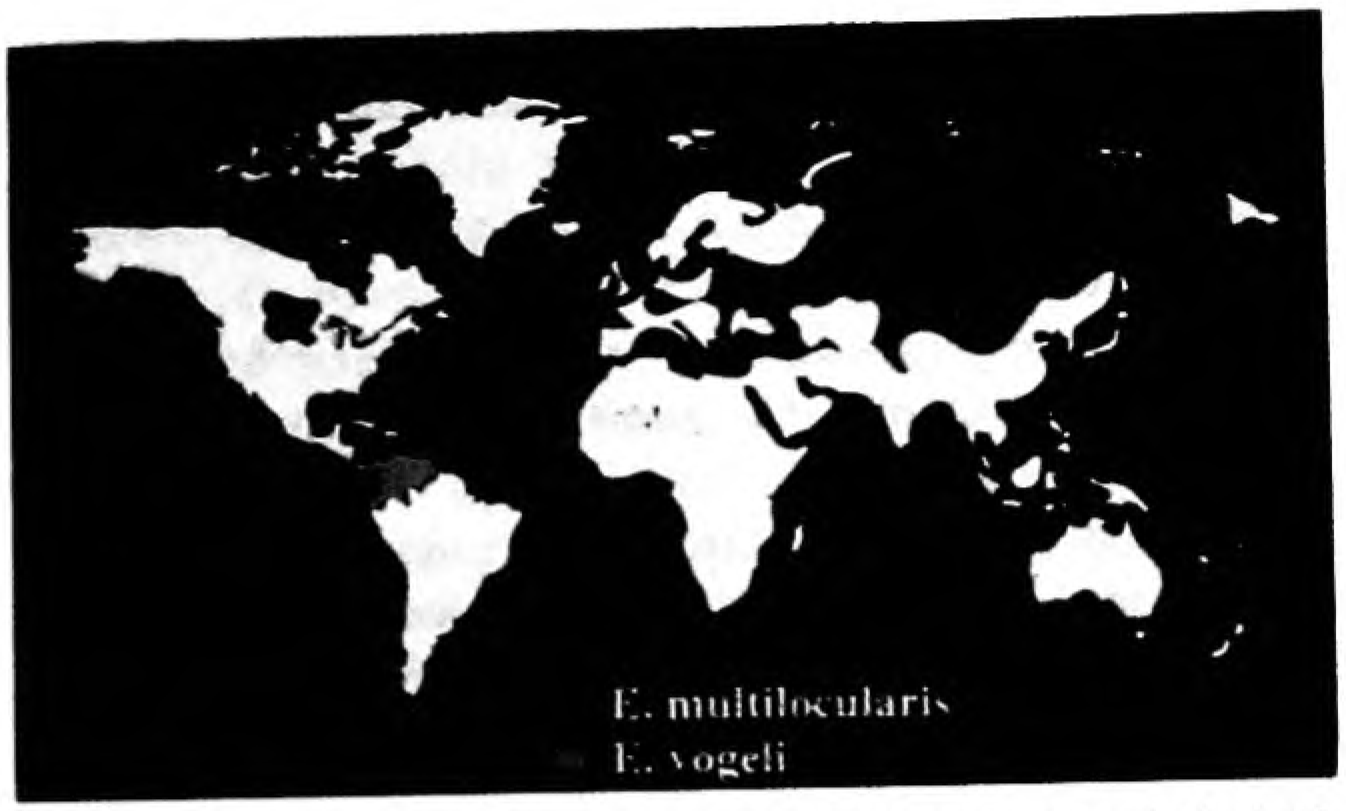

FIGURE 4. Geographical distribution of alveolar and polycystic hydatid disease.

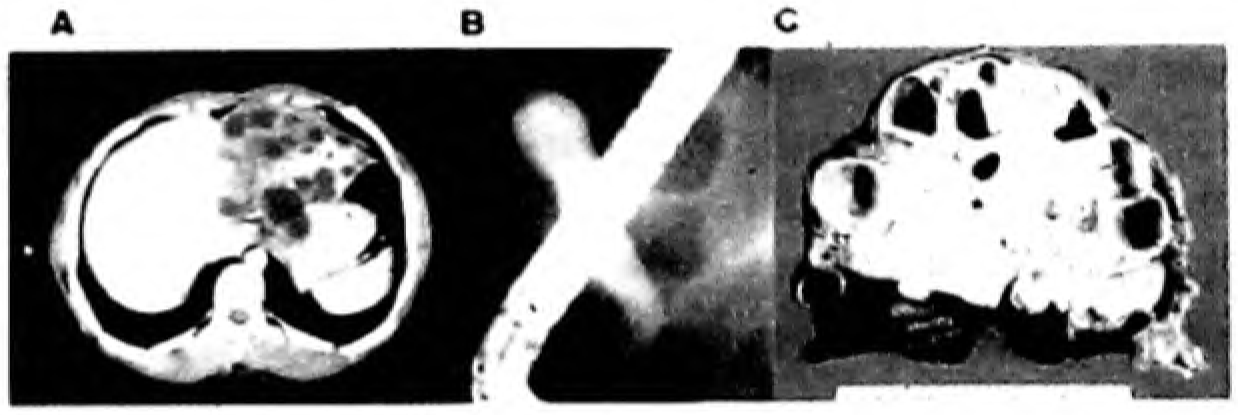

FIGURE 5. Polycystic hydatid disease with cholestasis. Panel A shows contrast-enhanced CT scan of upper abdomen. Left lobe liver is replaced by large multicystic mass with involvement of surrounding structures. Panel B shows a cholangiogram obtained at ERCP. Common bile duct in its upper part revealed a complete obstruction by infiltrating cyst. Panel C shows resected left lobe liver revealing multicystic mass. Each cyst was fluid filled with surrounding laminated membrane. The hooklets from the fluid had characteristic morphology of *Echinococcus vogeli*.

Alveolar Hydatic Disease

Geographic Distribution (Figure 4)

E. multilocularis has limited distribution in the northern hemisphere, within a belt stretching from the Northern Tundra zone southward to some regions around the 40th to 45th degree of northern latitude.[13] Human alveolar hydatid disease has been reported in central parts of Europe (Southern Germany, Western Austria, Switzerland and Eastern France), some parts of Russia (Siberia), Eastern Azerbaijan, some parts of Turkey (Eastern Anatolia), Northern Iran, Northern Afghanistan, Northern India (Kashmir), NOrthwestern Canada, Western Alaska (S

Lawarence Island) and Northern Japan (Hokkaido). The annual incidence of human infection is low (0.03 to 1.2 per 100,000 per annum) in most of the areas where disease is reported.[54-57]

Pathogenesis and Pathology

Humans are exposed to E. multilocularis by direct handling of the infected fox carcass or through ingestion of beries contaminated with cestode eggs. The egg hatches in the small intestines, release the oncosphere which penetrates the mucosa and is carried via blood or lymphatics to the liver. In the liver, metacestode divides asexually by lateral budding of germinal tissue and the secondary vesicles invade and infiltrate the surrounding host tissue. Surrounding each vesicle, there is intense host cell response consisting of epitheloid cells, lymphocytes and thick collagen tissue. This gives rise to a tumor, which may cavitate in the center. Calcification of the lesion is seen in over 70% of the cases and occurs as clusters of micro-calcifications or plaque-like calcific foci, with irregular distribution in central or peripheral parts of the lesion. In humans, the parasite does not form protoscolices and is made of a thin cuticle with non-germinative membrane demarcating a small cavity.

Primary infection occurs in the liver in all cases. The disease invades bile ducts, portal veins, hepatic veins and inferior vena cava and the adjacent structures beyond the organ borders. The disease metastasizes via the blood stream and lymphatics to distant organs namely lung, liver and bone. The growth of alveolar cysts in liver and other body organs is very slow and requires 5 to 15 years for the cyst to become symptomatic.[54]

Clinical Manifestations

The age of presentation of the alveolar disease is in 5th decade and beyond, with equal sex distribution. The disease has insidious onset and is detected as an incidental finding in over one-third of patients.[13] Hepatic disease presents as fatigue, epigastric pain, abdominal mass, and abnormal liver function tests. The disease has chronic progressive clinical course. OVer the years, patients devleop progressive hapatic failure (extensive hepatic infiltration), cholestatic jaundice (biliary obstruction), Budd-Chiari syndrome (hepatic venous outflow obstruction), protal hypertension (portal venous obstruction) and ascites (peritoneal involvement). Hepatic lesion can also grow into the pleura, lungs, inferior vena cava, right atrium, pericardium, retroperitoneum, kidneys and pancrease. This results in varied clincial manifestations depending on the organ involved. Apart from contiguous spread, the parasite metastasizes to the lungs, brain and bones resulting in multiple foci of Echinococcus infection. If untreated, the disease results in death in most of the patients in around 10-15 years.[54-59]

Diagnosis

The routine laboratory tests do not help in the diagnosis of alveolar hydatid disease. However, such tests help to assess the extent of hepatic involvement.

Serology

Serologic tests are very useful in diagnosis. Enzyme immunoassay using specific antigen Em2plus (mixture of purified metacestode antigen Em2 and a recombinant EmII/3-10 antigen) has a sensitivity of 97.1% and specificity of 98.9% in the diagnosis of alveolar hydatid disease.

Imaging

The parasite liver lesions have varied ultrasound patterns: solid heterogeneous echo texture with multiple echogenic nodules (hailstorm pattern), echogenic area associated with cystic zones (geographical map pattern) and transonic pseudocystic necrotic zone with debris. CT scan reveals heterogeneous hypodense mass, which does not enhance with intravenous contrast. Clustered micro-calcificaton and characteristic perinecrotic plaque-like calcification are detected in these lesions. Magnetic resonance imaging is very useful in assessing the spread of disease to hepatic veins, portal veins and bile ducts. ERCP and hepatic venography may be needed to find the site and extent of bile duct and hepatic vein disease, and for possible dilation and stenting.[56,57]

Treatment

Treatment options include redical resection of the involved liver segments or other affected organs, long-term chemotherapy and liver transplantation. Radical liver resection is considered in patients with localized liver disease without involvement of major blood vessels and distant metastasis. Around 20%-40% of patients at presentation are amenable to radical liver resection. Hemihepattectomy or extended (trisegment) liver resection can be performed with operative mortality of 0%-5%. Following radical surgery, chemotherapy is recommended for two years and disease surveillance for 10 years. Long-term survival in patients with radical resection is nearly 100%.[36,54]

Long-term (life-long) chemotherapy is indicated in patients with inoperable disease or in those with incomplete surgical resection. Benzimidazole derivatives (mebendazole or albendazole) are generally well tolerated, and adverse reactions are usually mild and transient. These drugs suppress metacestode growth and stabilize tumor size. Tumor regression occurs in around 20% of patients. Surveillance of patients on chemotherapy has shown marked improvement (10-year survival of 80%-83%, and a 15-year survial of 53%-83%) when compared to historical patients on no chemotherapy (10 year survival of 0%-25% and a 15-year survival of 0%).[13,36]

Liver transplantation has been performed in a small group of patients with extensive liver involvement and liver occurs in around half of patients within one year of transplant. Six-year suvival of 66% has been reported. In view of disease recurrence, adjuvant ost-transplant chemotherapy is recommended.[60]

Polycystic Hydatid Disease

Polycystic hydatid disease is the developed metacestode of E. vogeli or E. oligarthrus in humans. E. vogeli is a rare human disease, and sporadic human cases have been reported in neotropical America (Panama, Ecuador, Colombia, and Venezuela) and French Guiana (Suriname) (Figure 4). e. oligarthrus is an extremely rare human disease and only isolated case reports have been recorded from Venezulea, Brazil, India and suriname.[61-64]

Pathogenesis and Pathology

Bush dogs, the definitive hosts, play little role in direct exposure in humans. In contrast, hunting dogs fed on the raw viscera of pacas get infected and then expose humans. E. vogeli and E. oligarthrus metacestodes develop by endogenous proliferation of membranes in a unique fashion resulting in the formation of polycystic masses (Figures 5). Cysts of varying size are grouped together, each containing a yellowish fluid or gelatin-like substance. Cysts are often packed with proliferated laminated membranes that beome convoluted giving a cerebroid configuration. Brood capsules and protoscolices are formed within the cavities. Cysts are surrounded by a chronic inflammatory foreign body granulomatous reaction similar to that observed in parasite-induced granulomas. The larval structures often degenerate with secondary calcification. Calcification is seen in around half of cysts and may be annular, bizarre or amorphous.[61]

Liver is the primary site of infection in E. vogeli. Surrounding structures, like diaphragm lungs, pericardium, heart, mesentery and omentum, can be involved by contiguous spread. In contrast, E. oligarthrus produces polycystic disease in only extrahepatic tissue, namely orbit, lower jaw, heart and other sites without liver involvement.[58-61]

Clincial Manifestations

Polycystic hydatid disease of the liver presents in the fourth decade and beyond. The majority of patients present with abdominal pain, abdominal distension, fever and weight loss. Clinical iexamination reveals hepatomegaly and hard, round masses on the liver surface. The disease is slowly progressive and clinical course is complicated by progressive liver failure, cholestasis, portal hypertension and involvement of surrounding structures.[58]

Diagnosis

Serologic tests are positive in most of the patients, especially in those with hepatic involvement. E. vogeli and E. oligarthrus share antigens with other Echinococcus species, thus current immunodiagnostic tests do not permit specific diagnosis of polycystic disease. Ultrasound, CT scan and MRI are useful in defining polycystic large fluid-filled larval cestode lesions with diffuse calcification. However, specific or characteristic findings of these cysts have not been reported in detail. Aspiration of the cysts, espeically in the extrahepatic sites, is recommended and shows scoleces. Species diagnosis and differentiation is possible by examining the sizer and shape of rosteller hooklets in the scoleces. Anaphylaxis has been reported with aspiration of soft-tissue cysts, and needs to be done under close patient monitoring.[61-64]

Treatment

Radical surgical excision of cysts in the soft tissue, if possible, is the ideal treatment. Albendazole therapy prior to and following excisiion is recommended, particularly if the excision is not complete. Liver cysts need radical excision if localized. Extensive disease can be treated by long-term albendazole and praziquantel therapy.[61-64]

References:

1. Pawlowski ZS. Terminology related to echinococcus and echinococcosis. Acta Trop 1997;67:1-5.
2. Lewall DB. Hydatid disease: biology, imaging and classification. Clin Radiol 1998;53:863-74.
3. Arambulo P. Public health importance of cystic echinococcosis in Latin America. Acta Trop 1997;67:113-24.
4. Altintas N. Cystic and alveolar echinococcosis in Turkey. Ann Trop Med parsitol 1998;92:637-42.
5. Chai JJ. Epidemiological studies on cystic echinococcosis in China. Biomed Environ Sci 1995;8:112-36.
6. Euzeby J. The Epidemiology f hydatidosis with reference to the Mediterranean area. Parasitologica 1991:33:25-39.
7. Eckert J, Thompson RC. Echinococcus strains in Europe. Trop Med Parasitol 1998;39:1-8.
8. Jenkins DJ. Hydatidosis: a zoonosis of unrecognized increasing importance? J Med Microbiol 1998;47:281-2.
9. Helbig M, Frosch P, Kern P,Frosch M. Serological differentiation between cystic and alveolar echinococcosis by use of recombinant larval antigens. J. Clin Microbiol 1993;31:3211-5.
10. Karavias DD, Vagianos CE, Bouboulis N, rathosis S, Androulakis J. Improved techniques in the surgical treatment of heptic hydatidosis. Surg Gynecol Obstet 1992;174:176-80.
11. Dykes PW, Marriner S, et al. Albendazole: objective evidence of response in human hydatid disease. JAMA 1985;253:2053-7.
12. Khuroo MS, Wani NA, Javid G, et al. Percutaneous drinage compared with surgery for hepatic hydatid cysts. N Engl J Med 1997;337:881-7.
13. Ammann RW, Eckert J. Cestodes: Echinococcosis. Gastroenterol Clin North Am 1996;25;655-89.
14. Elton C, Lewis M, Jourdan MH. Unusual site of hydatid disease Lancet 2000;355:2132.

15. Milicevic M. Hydatid disease. In: Blumgart LH, editor. Surgery of the liver and biliary tract. 2nd edition. Vol. 2, Edinburgh: Churchill Livingstone. 1994;1121-50.
16. Thomas PS, OWen ER, Larson HE. Hydatid disease and cholestasis. Br J Clin Pract 1990;44:336-7.
17. Zanzi I, Sapunar J, Croizet V, Bancalari G. Budd-Chiari syndrome due to hepatic hydatidosis. Boletin Chileno de Parasitologica 1967;22:113-8.
18. Zargar SA, Khuroo MS, Khan BA, Dar MY, Alai MS, Koulx P. Intrabiliary rupture of hydatid cyst: sonographic and cholangiographic appearances. Gastrointest Radiol 1992;17:41-5.
19. Gielekas D, Papacosta D, Papaconstantinou C, et al. recurrent anaphylactic shock as a manifestation of echinococcosis. Scand J Thorac Cardiovasc 1986;20:175.
20. Werczberger Gohlman J, Wertheim G, Gunders AE, et al. Disseminated echinococcosis with repeated anaphylactic shock treated with mebendazole. Chest 1979;76:482.
21. Khuroo MS. Percutaneous drainage of hydatid cysts. N Engl J Med. 1998;392:391-3.
22. Merkle EM, Schulte M, Vogel J, Tomczak R, Rieber A, Kern P, Goerich J, Brambs HJ, Musculoskeletal involvement in cystic echinococcosis: report of eight cases and review fo the literature. Am J Roentgenol 1997;168:1531-4.
23. Yatto GN, Khuroo MS, Zargar SA, Bhat FA, Sofi BA. Percutaneous drinage of the hydatid pancreatic cyst with obstructive jaundice. Journal of Gastroenterology & Hepatology 1999;14:931-4.
24. Hegeman G, Gottstein B, Witte OW. Isolated Echinococcus granulosus hydatid cyst in the CNS with severe reaction to treatment. Neurology 1999;52:1100-1.
25. Craig PS. Detection of specific circulating antigen, immune complex and antibodies in human hydatidosis from Turkana (Kenya) and Great Britain, by enzyme-immunassay. Parasite Immunol 1986;8:171-88.
26. Wattal C, Malla N, Khan IA, Agarwal SC. Comparative evaluation of enzyme-linked immunosorbent assay for the diagnosis of pulmonary echinococcosis. J Clin Microbiol 1986;24:41-6.
27. Lightowlers MW, Gottstein B. Echinococcosis/hydatidosis: antigens, immunological and molecular diagnosis. In: Thompson RCA, Lymbery AJ, editors. Echinococcosis and Hydatid disease. Wallingford, oxon: CAB International 1995;355.
28. McManus DP. Molecular technology: improving strategies for controlling hydatid disease and cysticercosis. Southeast Asian J Trop Med public Heatlh 1990;21:161-173.
29. Von Sinner W. Advanced medical imaging and treatment of human cystic echinococcosis. Semin Roentgenol 1997;32:276-90.
30. Jerray M, Benzarti M, Garrouche A, et al. Hydatid disease of lungs: a study of 386 cases. Am Rev Respir Dis 1992;146:185.
31. Reeder MM, Palmer PES. The Radiology of Tropical Diseases with Epidemiological, Pathological and Clinical Correlation. Baltimore: Williams & Wilkins, 1981.
32. Gori S, Campetelli A, Luchi S, Paladini A, Savalli E, Scasso A. Cytology in the percutaneous treatment of hydatid cysts: a report of four cases. Acta Cytol 1993;37:423-6.
33. Ekrami Y. Surgical treatment of hydatid disease of the liver. Arch Surg 1976;111:1350-2.
34. Langer JC, Rose DB, Keystone JS, Taylor BR, Langer B. Diagnosis and management of hydatid disease of the liver: a 15-year North American experience. Ann Surg 1984;199:412-7.
35. Sayek I, Yalin R, Sanac Y. Surgical treatment of hydatid disease of the liver. Arch Surg 1980;115:847-50.
36. WHO Informal Working Group on Echinococcosis. Guidelines for treatment of cysic and alveolar Echinococcosis in humans. Bull WHO 1996;74:231-42.
37. Morris DL. Echinococcous of the liver. Gut 1994;35:1517-8.
38. Gil-Grande LA, Boixeda D, Garcia-Hoz F, et al. Treatment of liver hydatid disease with mebendazole: a prospective study of thirteen cases. Am J gastroenterol 1983;78:584-8.
39. Gil-Grande LA, Rodriguez-Caaberio F, Prieto JG, et al. Randomized controlled trial of efficacy of albendazole in intra-abdominal hydatid disease. Lancet 1993;342-1269-72.
40. Teggi A, Lastilla MG, De Rosa F. Therapy of human hydatid disease with mebendazole and albendazole. Antimicrob Agents Chemother 1993;37:1679-84.
41. Mohammad AE, Yasawy MI, Karawi MA. Combined albendazole and praziquantel versus albendazole alone in the treatment of hydatid disease. Hepato-Gastroenterol 1998;45:1690-4.
42. Filice C, Brunetti E, Bruno R, Crippa FG. Percutaneous drinage of echinococcal cysts (PAIR-puncture, aspriation, injection, reaspiration): results of a worldwide survey for assessment of its safety and efficacy. WHO Informal Working Group on Echinococcosis. PAIR Network. Gut 2000;47:156-7.
43. Filice C, Pirola F, Brunetti E, Daughetti S, Strosselli M, Foglieni CS. A new therapeutic approach for hydatid liver custs: aspriation and alcohol injection under sonographic guidance. Gastroenterology 1990;98:1366-8.
44. Khuroo MS, Zargar SA, Jahajan R. echinococcus granulosus cysts in the liver: management with percutaneous drainage. radiology 1991;180:141-5.
45. Khuroo MS, Dar MY, Yattoo GN, et al Percutaneous drainageversus albendazole therapy in hepatic hydatidosis: a prospective randomized study. Gastroenterolgy 1993;104:1452-9.
46. Gargouri M, Ben Zmor N, Ben Chehida F, et al. Percutaneous treatment of hydatid cysts (Echinococcus granulosis). Cardiovasc Intervent Radiol 1990;13:169-73.
47. Akhan O, Ozman MN, Dincer A, Sayek IS, Gocvmen A. Liver hydatid disease: long term results of percutaneous treatment. Radiology 1996;198:259-64.
48. Brunetti E, Filice C. Percutaneous aspiration in the treatment of hydatid liver cysts. Gut 1996;38:936.
49. Castellano G, Moreno-Sanchez D, Guiterrez J, Moreno-Gonzalez E, Golina F, Solis-Herruzo JA. Caustic sclerosing cholangitis: report of four cases and a cumulative review of the literature. Hepatogastroentrology 1994;41:458-70.
50. Tsimoyiannis EC, Grantzis E, Moutesidou K, Lekkas ET. Secondary sclerosing cholangitis: after injection of formaldehyde into hydatid cyst in the liver. Eur J Surg 1995;161:299-300.
51. Al Karawi MA, Yasawy MI, Mohammad AR. Endoscopic management of biliary hydatid disease: report on six cases. Econscopy 1991;23:278-81.
52. Lujan Mompean JA, Parcio PP, Campos RR, Ayllon JG. Laparoscopic treatment of a liver hydatid cyst. Brit J Surg 1993;80:907-8.
53. Moreno-Gonazalez E, Segurola CL, Urena MAG, et al. Liver transplantation for Echinococcus granulosus hydatid disease. transplantation 1994;58.
54. Wilson JF, Rausch RL. Alveolar hydatid disease: A review of clinical features of 33 indigenous cases of Echinococcus multilocularis infection in Alaskan Eskimos. Am J Trop Med Hyg 1980;29:1340-55.
55. Khuroo MS, Datta DV, Koshy A, Mitra SK, Chhuttani PN.

Alveolar hydatid disease of the liver with Budd-Chiari syndrome. postgrad Med. J 1980;56:197-201.

56. Choji K, Fujita N, Chen M, Spiers ASD, Morita Y, Shinohara M, Nojima T, Irie G. Alveolar hydatid of the liver. Computed tomography and transabdominal ultrasound with hisopathological correlation. Clin Radiol 1992;46:97-103.
57. Didier D, Wieler S, Rohmer P, Lassegue A, Deschamps JP, Vuitton D, et al. Hepatic alveolar echinococcosis: correlative US and CT study. Radiology 1985;154:179-86.
58. Tunaci M, Tunaci A, Engin G, Ozkorkmaz B, Ahishali B, Rozanes I. MRI of cerebral alvolar chinococcosis. Neuroadiology 1999;41:844-6.
59. Samuels S, Fosmoe R. Alveolar hydatid disease with involvement of the inferior vena cava. Am Surg 1970;36:698-701.
60. Bresson-Hadni S, Miguet JP, Mantion G, et al. Orthotopic liver transplantation for incurable alveolar echinococcosis of the liver report of 17 cases. Hepatology 1991;13:1061-70.
61. D'Alessandro A. Polycystic echinococcosis in tropical\America Echinococcus vogeli and Echinococcus oligarthrus. Acta Tropica 1997;67:43-65.
62. D'Alessandro A, Rausch RL, Cuello C, Aristizabal N. Echinococcus vogeli in man, with a review of polycystic hydatid disease in Columbia and neighboring countries. Am J Trop Med Hyg 1979;28:303-17.
63. Basset D, Girou C, Nizios IP, D'Hermies FD, Hoang C, Gordon R, D'Alessandro A. Neotropical echinococcosis in Suriname. Echinococcus oligarthrus in the orbit and Echinococcus vogeli in the abdomen. Am J Trop Med Hyg 1998;59:787-90.
64. Kini U, Shariff S, Nirmal V. Aspiration cytology of Echinococcus oligarthrus: a case report. Acta Cytol 1997;41:544-8.

CONFERENCE REPORT

"LAPARO UPDATE 2002"

Inaugration. Stiting for L to R: Prof. Muneer Khan (Organising Secretary) Mr. M.L. Kadalbaju(Commissioner Secretary H&ME) Mr. I Malhi (Chief Secretary) Dr. Parveen Kour (Principal GMC) Dr. Nazi Salroo (Joint Organising Secretary is Standing)

"Laparo Update 2002" an international conference and live Laparoscopic Workshop, was jointly organized by Govt. Medical College, Srinagar and MCI on the 25th and 26th of October 2002 at SKICC and SMHS Hospital, Srinagar. The Programme included a CME/Paper Presentation on the 25th and a Live Operative Workshop on the 26th October. Over 350 delegates of National and International repute took part in the conference. There was a distinguished faculty of surgeons from both India and abroad who took part in the demonstration of various Laparoscopic procedures. It was the first ever such type of conference to be held in the valley.

The Conference was inagurated on the 25th October by the Chief Guest, Mr. I.S. Malhi, the then Chief Secretary to Govt. of J&K at 10 a.m. This was followed by the Welcome address by the Organising Sect. Dr. Muneer Khan, Prof. and Head, Deptt. of Surgery, G.M.C., Srinagar. The Chairperson, Prof. Praveen Kour, Principal G.M.C., Srinagar also addressed the gathering. Among other eminent dignitaries who also spoke on the occasion were the Guest of Honour, Mr. H.L. Kadalbaju, the then Commissioner / Sect. Health and Medical Education Dept., Govt. of J&K; Dr. Mustafa Kamal, Ex-Minister for Health and Medical Education Dept., Govt. of J&K Dr. Nazir Ahmad Salroo, joint organising Sect. delivered the vote of thanks. Following that, the "Souvenir" was released by Mr. H.L. Kadalbaju. Dr. Mustafa Kamal inaugrated the "Scientific Exhibition" which included stalls of various Pharmaceutical companies and Laparoscopic/General Surgical equipment. A tea/refreshment break preceded the session of Guest Lectures.

Among the various Guest Lectures/Video Presentations delivered on the occasion were:

1. Role of Laparoscopy In Acute Abdominal Surgeries. Dr. R. Sarangi, Consultant Sir Ganga Ram Hospital.
2. Laparoscopy from candlelight to Sunlight. Dr. G.R. Mufti, Consultant Urologist & Director and Chairman, Medway, U.K.
3. Role of laparosocopy in Colorectal Cancer. Dr. Deep Goel, Consultant Sir Ganga Ram Hospital.
4. Laparoscopic treatment of Aortic Aneurysm. Dr. Ashok Gupta, Consultant Sir Ganga Ram Hospital.
5. Instrumentation in Laparoscopic Surgery with special reference o Lap. Ureterolithotomy. Dr. Sameer Rai- Consultant Urologist, Prem Rai Hospital, Amritsar.
6. Personal experience from Open to Laparoscopic surgery - Dr. Sheetal Singh; ex-H.O.D. Surgery, G.M.C. Jammu & Ex-Principal G.M.C. Srinagar.

This was followed by Lunch Break after which the Scientific Session started. Some of the Papers/Video presentations that were presented during this session were:

1. Dr. Deept Goel - Hand Assisted Laparoscopic Surgery.
2. Prof. Muneer Khan - Lap. Ureterolithotomy.
3. Dr. Sheikh Adil Bashir - Laparoscopic Treatment of Hydatid Cyst Liver.
4. Dr. Farhan Khan - Laparoscopic Retroperitoneal Nephrectomy.
5. Prof. N.A. Wani - Biliary Injuries in Lap. Cholecystectomy.
6. Dr. Shahida Massarat - Lap. Assisted Vaginal Hysterectomy.
7. Dr. Nazir A. Salroo - Lap. Appendectomy for acute appendicitis.
8. Dr. G.M. Sheikh - Adv. of Lap. Surgery.
9. Dr. Ajaz A. Rather - Complications of Lap. Cholecystectomy.
10. Dr. M.A. Wani and Dr. Yousuf Tak - Anesthetic considerations in Lap. Srugery.
11. Dr. Nisar A. Wani - Role of Laparoscopy in Oncology.
12. Dr. Manzoor Qazi - laser in Arthroscopic Surgery.
13. Prof. Gh. Jeelani and Dr. N.A. Khan - Management of Bladder tumors in Kashmir.

This was followed by a cultural programme organized by the students of G.M.C. Srinagar and an elaborate dinner at SKICC, Srinagar.

On the Morning of 26th October, a live operative workshop of laparoscopic surgery was held with a two way audio-visual system between the auditorium and the operating theatre. The following procedures were demonstrated.

1. Lap. repair of Inguinal hernia - Dr. R. Sarangi, Consultant, Minimal Access Surgery, Sir Ganga Ram Hospital, New Delhi.
2. Lap. Spleenectomy - Dr. Deep Goel. Consultant Laparo Surgeon and Colorectal Surgeon, Sir Ganga Ram Hospital, New Delhi.
3. Lap. Cholecystectomy with CBD exploration- Dr. R. Sarangi.
4. Lap. repair of incisional hernia - Dr. R. Sarangi.
5. Lap. Ureterolithotomy - Dr. Deep Goel.
6. Lap. Assisted Vaginal Hysterectomy - Dr. Shahida Massarat, Consultant Gaynecologist LDH, Srinagar.
7. Lap. Nephrectomy - Dr. R. Sarangi.

The workshop continued till 6 p.m. after which a dinner was held at Hotel Grand Palace International.

This live international workshop was the first of its kind to be held in J&K Stateand was of tremendous benefit to young surgeons and medical students who witnessed these laparoscopic procedures for the first time.

Dr. N. A. Salroo

XVI ANNUAL NORTH ZONE OPHTHALMOLOGICAL SOCIETY CONFERENCE

NORTH ZONE OPHTHALMOLOGICAL SOCIETY
XVI ANNUAL CONFERENCE
OCT 26 TH 27 TH'02

North Zone Ophthalmological Society comprising of the States of Himachal Pradesh, Jammu and Kashmir, Punjab, Haryana and Union Territory of Chandigarh was established in 1987; Medical College Jammu had the distinction of holding the Ist conference of the society. Annual conferences of the society has been held since then by each State in rotation. Two more conferences of the NZOS the 6th and 11th in the year 1992 ad 1997 were also held at Medical College, Jammu.

XVI Annual Conference of the North Zone ophthalmological society was held on 26th and 27th October this year at a new venue, the upcoming complex of Acharya Shri Chander College of Medical Sciences, Sidhra, Jammu. The conference was inaugurated by Prof. R.R. Sharma, Vice Chancellor Jammu University. Dr. S.S. Soodan, Prof. and H.O.D. Ophthalmology, Acharya Shri Chander College of Medical Sciences, Sidhra, Jammu, took over as president of North Zone Ophthalmological Society. For the first time seven leading NGO's engaged in the field of eye care in the state were honoured at the conference. More than two hundred delegates attended the conference. One hundred twenty four delegates had registered from outside the State of Jammu and Kashmir, seventy four delegates had registered from within the state. Conference programme also included a trade exhibition. In spite of difficult situation and militancy 26 pharmaceutical, surgical and non-surgical concerns took part in the exhibition.

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This year's conference had exciting programme in the form of free papers, symposium guest lectures and instruction courses. There were fifty papers which covered different aspects of ophthalmology. The symposium on complications of Intraocular surgery was covered by eminent ophthalmologists of the region in about 20 papers.

Guest lecturers were delivered by Dr. Atual Kumar from A.I.I.M.S. New Delhi and Dr. N. Shroff form Shroff Eye Hospital, New Delhi. Dr. Atual Kumar delibereated on (1) Indocyanine Green and Flourescien Fundus Angiography imaging in Age Related Macular Disease, (2) Posterior Viterous Retraction-recent advances in management. Dr. N. Shroff delved on Phacoemulsification in difficult situations. Delegates showed keen interest and appreciated the guest lecturers. Instruction courses were held on ultrasonography in ophthalmology and sterilization and good O.T. practices. Delegates appreciated the quality & quantity of Scientific feast served to them at the conference. The conference ended with a vote of thanks to the management of Acharya Shri Chander College of Medical Sciences for the help and facilities provided for organisation of the conference.

Dr. Madan Gopal Gupta
Organising Secretary,

Dr. Guran Ditta Mal
Eye Clinic,
Kachi Chawni, Jammu.

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Consolidated Cybernetics Co. Pvt. Ltd., now has the distinction of having both its Quality Management System (ISO 9001 : 2000) and product certified. With this, the Company expects to surge ahead in the Health Care Management area both in the Indian and global markets. The Company is also participated in HOSPI MEDICA ASIA 2002, during 10th - 12th September 2002 at Singapore, the largest exhibition of its kind for the Health Care Industry.

P.R. Rangaswami
Management Director

CONDOLENCE

A condolence meeting of the Departments of Pathology, Microbiology, and Medicine Govt. Medical College, Srinagar was held to pay tributes to Dr. Santosh Kapoor (Sindhwani) Ex. Associate Prof. Department of Pathology GMC, Srinagar on hearing the sad and unbelievable news of her untimely death. The efficiency, dedication and honesty of the doctor will always be remembered. The gathering prayed for the departed soul.

A similar tribute was also paid by the Executive Council of Medical faculty association (MFA) Govt. Medical College Srinagar. In the condolence meeting held on 20-11-2002 all the members expressed their sympathy with the bereaved family and prayed for the departed soul.

The Editorial Board of JK-Practitioner are also shocked by her untimely death and expressed their sympathies to Dr. Manhoar Lal Sindhwani and their children Maneesha and Sumaidha and pray to God give them courage to bear the irreparable loss.

Dr. G.M. Malik
President IMA
Kashmir Branch

Dr. M.L. Bhat
Hed Deptt of
Pathology GMC
Srinagar

Dr. Mir Nazir Ahmad
President
MFA, Srinagar

Announcement:

Medicine Update: (International Conference on Recent Advances in Medicine) is being held from 18th-20th July 2003 in which a galaxy of overseas experts from USA / UK / Middle East and renowned experts in medicine within the country are expected to participate. For details contact:

Overseas delegate: faroquekhan@yahoo.com.
Indian delegate: Prof. G.Q. Khan, HOD Medicine
Organizing Secretary
SMHS Hospital Srinagar
Tele No's (O) 0194-2452013
Ext. 465
(R) 0194-2430530
E-mail: gqk@rediffmail.com., akmaliyah@hotmail.com

Islamic Medical Association of North America

IMANA Goes to China in 2003

The 36th Annual and 7th International IMANA Convention will be held Insha'Allah in Beijing, China from July 24 to July 30, 2003.

Due to the great interest in the Islamic history of China, the organizers anticipate a very large number of participants. Therefore, participants will be divided into two major groups. The first group, Group A, will arrive in Shanghai on July 16, take tours of four cities, Shanghai, Lanzhou, Xian and Guilin, and reach Beijing to attend the conference before leaving for home on July 31. The second group, Group B, will arrive in Beijing on July 24, attend the conference through to July 30 and then leave for the tour of the four cities and return home from Shanghai on August 8. There will be a stay of six nights in Beijing and nine nights in other cities in total.

Source: IMANA Newsletter Fall 2002

RAM NATH CHOPRA (17.8.1882-13.6.1973)

Ram Nath Chopra regarded as the father of Pharmacology in India was born on August, 1882 in a Punjab family, domiciled in the State of Jammu & Kashmir. In 1903 he joined the University of Cambridge for a degree in medicine. There at a latter date he came under the influence of Dr. Walter E Dixon the first professor of pharmacology at Cambridge. It is this influence which shaped Ram Nath Chopra into a keen Experimental Pharmacologist. In 1908 he was awarded a degree of MD (Medicine) for his Original experimental work related to the action of various drugs on ciliary movements.

50
BOMBAY
17-8-83
RAM NATH CHOPRA
RAM NATH CHOPRA

On his return to India he was required to do military duty as an officer in the Indian Medical Service. It was after about a decade that an oppournity came for him to impart his skills to others. In 1921 he was appointed Professor of Pharmacology in the newly established Calcutta School of Tropical Medicine. At the same time he was also appointed to the chair of Pharmacology at the Calcutta Medical School. This dual responsibility not only fulfilled his appetite for research but also realized his ambition to follow in the footsteps of Prof. Dixon. He was the first in India to give lectures/demonstrations to undergraduate medical students. At the Tropical School of medicine he would go into extensive investigations into the merits and demerits of Indian folklore medicine.

This dual role ,as he himself points in his writings afforded wonderful opportunities of putting Pharmacology on the "Medical Map" of India. It was not an easy task to transform Pharmacology into an independent discipline from the "materia medica" taught by professor of clinical medicine at a hospital. By the time he retired from active Indian Medical Service in 1941 most of the medical schools in India had established independent chairs in Pharmacology.

Among his many publications the following are considered reference works of great signifiance-1. Indigenous drugs of India 2. Glossary of Indian Medicinal Plants 3. Poisonous Plants of India 4. Drug addiction in India.

Several honours and awards were conferred on RN Chopra in recognition of his outstanding contributions to Indian Pharmacology. In 1953, he was appointed Honorary Physician to King (George V) and was promoted Colonel with Brevet rank. In 1938 he was elected fellow of the Royal College of Physicians (FRCP), London and was knighted by the British Government in 1941. He was appointed president of National Institute of Science in 1939. He was also a recipient of the Minto Gold Medal (1925), CIE (1934) and the Squibb Award. He was appointed the Director of the Drug Research Laboratory at Srinagar between 1941 and 1957 and Honorary Scientific Advisor to the Regional Research Laboratory, Jammu from 1958 onwards.

Dr. Chopra was well known for his humility, courtesy and kindness. He was never at a loss to recite an Urdu or a Persian couplet appropriate to the occasion. He died in Kashmir on June 13, 1973. Indian Posts and Telegraphs Department issued a Commemorative Postage stamp in honour of Brevet Colonel Sir Ram Nath Chopra on his 110th. Birth anniversary on August, 1983.

Shafiqa Aslam & Zaffar Abbas

Good News for Doctors Preparing for Post-graduate entrance examination (M.D., M.S., etc.)

The institute of advance medical studies (AIMS) New Delhi has authorised us to open a booking centre for postal coaching for post graduate entrance examination in medicine. At present three different programmes run by them are:

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(Near Dr. GM Malik's Clinic) 8 a.m. to 11 a.m.
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Suggested topics:-

1.Emerging worldwide antibiotic resistance. Implications for Kashmir with suggested remedies.

OR

2.Medical Ethics: Should physicians receive grants, gifts, sponsorships for conference from pharmaceutical industries.

Prof.GM Malik
Chairman Academic Committee
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REVIEW

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GASTROINTESTINAL AND HEPATOBILIARY MICROSPORIDIOSIS

Rafiq A. Sheikh, MBBS, MD, MRCP (UK), FACP, FACG; Shagufta Yasmeen, MD, MRCOG; Thomas Prindiville, MD

INTRODUCTION

Although more than two thirds of patients with acquired immunodeficiency syndrome (AIDS) have abnormal liver function tests and hepatomegaly, only a subset of these patients have disease of the biliary system (1, 2, 3). Several biliary tract abnormalities have been reported in patients with HIV infection. Most of them are related to opportunistic infections peculiar to an immunodeficient state and similar diseases have been reported in immunodeficient states other than AIDS. The biliary tract may also be involved in other diseases associated with the acquired immunodeficiency syndrome like Kaposi's sarcoma and lymphoma. Common biliary tract diseases in patients with AIDS include papillary stenosis, acalculous cholecystitis and intrahepatic and/ or extrahepatic sclerosing cholangitis. In 1989, Cello (4) described in detail the spectrum of cholangiographic abnormalities in acquired immmunodeficiency syndrome now referred to as, "AIDS cholangiopathy" and additionally reported on short and long term benefit of sphincterotomy

The AIDS related Cholangitidies are believed to be caused by opportunistic infections. Initial reports of cholangitis in AIDS patients described an association with cryptosporidium and cytomegalovirus infections(5,6,7). Cryptosporidium and cytomegalovirus were the inceptive pathogens described in relation to AIDS cholangiopathy. Even though other pathogens are being increasingly reported, cryptosporidium remains to be the most common causative pathogen in AIDS related cholangiopathy and is identified in the bile duct epithelium or stools in 20% to 62% of patients(4, 8). Cytomegalovirus is the second most common cause of AIDS related cholangitis and has been described in 23 % to 42 % of patients.

In 1983, Pitlik, et al (6) and Guarda, et al(5) reported extrahepatic biliary obstruction from opportunistic infection by Cryptosporidium in patients with AIDS. Subsequently, acalculous cholecystitis(9), sclerosing cholangitis(7) and paillary stenosis (10) were also described in patients with AIDS in association with opportunistic infections by Cryptosporidium and/ or cytomegalovirus.

Microsporidia are a less common etiology of AIDS related cholangitis and account for about 10% cases[11]. Enterocytozoon beinesui is the most common intestinal microsporidium encountered in patients with AIDS and accounts for most of the cases of sclerosing cholangitis(1). In 40% to 50% cases with AIDS related cholangiopathy , no opportunistic microorganisms are found(10, 12). It is likely that many of these cases with AIDS related cholangitis without an identifiable pathogen may have microsporidiosis(1). Pol, et al(10) reported a series of 8 patients with AIDS related cholangitis who had negative routine workup for opportunistic infections and were found to have microsporiodosis on electron microscopy.

E. intestinalis is a recently described microsporidia species first reported by Orenstein et al, in association with chronic diarrhoea in AIDS patients(13). It accounts for about 10 % cases of intestinal microsporidiosis(14) and responds to albendazole . A unique feature of this species is that it infects not only enterocytes but also infects the lamina propria macrophages, fibroblasts and endothelial cells(15, 18,19). It has a tendency to disseminate to distant sites and has been recovered from colon, liver, gall bladder, urinary tract and respiratory tract of infected patients(20). Encephalitozoon (septata) intestinalis is the second most common intestinal microsporidium but appears to be an uncommon cause of AIDS related cholangiopathy, only few cases having been reported(15,19,20).

Mycobacterium avium intracellulare (MAI) and isospora belli are uncommon causes of AIDS related cholangitis and occur in less than 5% patients(4,8). Lymphoma and Kaposi's sarcoma very rarely cause a picture of cholangitis in patients with AIDS(4).

DISCUSSION

EPIDEMIOLOGY

Microsporidia are ubiquitous, sporulating, obligate intracellular protozoa belonging to the phylum Microspora These parasites were first described in silkworms in 1857(20) and are known to infect vertebrates, invertebrates and humans. Out of approximately 100 genera and more than 1,000 species of microsporidia described(21), at least ten species are known to infect humans(20,22). The first case of humans microsporidial infection was reported in 1959 (23). Microsporidial infections in immunocompetent subjects are

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rare and only 10 cases of well documented infections have been reported in HIV negative patients, three of them being imunosuppressed for other reasons(24). In 1985 a new species of microsporidia, Enterocytozoon bieneusi (E. bieneusi) was described in AIDS patients with diarrhoea(25). Several hundred cases of human microsporidiosis have been described since then and the most prevalent species are E. bieneusi and E. intestinalis(26, 27). These two species have only been identified in humans and have been reported from Europe, North and South America and Africa (26, 27). The prevalence rates of microsporidia in patients with AIDS and chronic diarrhoea range from 7- 50%(22) and accounting for 10- 30% of diarrhoeal diseases in these patient(20). Although majority of the AIDS patients with microsporidiosis present with intestinal involvement, disseminated infections are increasingly recognized.

Microsporidia have been identified in the gall bladder and biliary tree of AIDS patients suggesting their role in cholecystitis and cholangitis. The spectrum of disease associated with microsporidiosis include keratoconjunctivitis, sinusitis, bronchiolitis, pneumonitis, nephritis, cystitis, prostatitis, hepatitis, peritonitis and myositis (15,17,25,27,28).

MORPHOLOGY AND CELL BIOLOGY

Five genera of microsporidia are known to cause disease in humans, viz, Enterocytozoon, Septata, Nosema, Pleistophora and Encephalitozoon(21,27,29). Septata intestinalis has been reclassified in the genus Encephalitozoon. Enterocytozoon bieneusi, first described in 1985, is responsible for 90% of human microsporidial infections(20, 23).

Microsporidia are intracellular parasites with small 1-5 µm infective spores capable of cell invasion. Mature spores contain a polar filament which distinguishes them from other organisms. The exact mode of transmission of microsporidia is not well known, although person-to-person transmission by fecal-oral route is suspected(25,26). Infections are believed to occur from ingestion or inhalation of spores from contaminated urine, stools and possibly respiratory secretions. The spores germinate in the epithelial cells of the gastrointestinal or the respiratory tract. During germination the nuclear material from the sporoplasm is propelled into the polar filament which is then used to inject the sporoplasm into the host cell. Spores may directly be released from the infected enterocytes into the lumen or parasite laden enterocytes may be shed into the lumen. The spores may remain viable for many years.

There are some distinct differences between the cell biologies of the two most common microsporidia. E. bieneusi commonly found in and first described in intestinal enterocytes, has also been reported in bile duct epithelial cells, pancreatic duct, gall bladder, liver, nose, trachea and bronchus. The organism replicates mainly in the jejunal enterocytes and perhaps also in the duodenal enterocytes. The infected villi typically become shorter or broader but may look histologically unremarkable in some cases. Spores are seen in stools but have not been reported in urine or respiratory secretions(10).

E. intestinalis also develops in the host cell and infects cells down to the crypt area including the lamina propria macrophages. This is a characterestic feature of E. intestinalis although recently also reported in E. bieneusi(30). E. intestinalis has a single nucleus and 2- 1.2µm spores with 4- 7 polar filamentous coils. Spores may be more clustered than E. bieneusi. In contrast with E. bieneusi, these organisms are found in the stools, urine, alveolar macrophages, endothelial cells and Kupffer cells of liver(16,29). This widespread distribution accounts for the disseminated infections in the immunocompromised host.

CLINICAL FEATURES

The first case of human microsporidiosis from Encephalitozoon like organisms was reported in 1959 in a Japanese boy who presented with seizures(21). Since the advent of AIDS epidemic in 1980s, microsporidial infections are being increasingly reported in immunosuppressed patients(27). Majority of HIV-infected patients present with enteritis, although involvement of other organ systems has been reported.

Gastrointestinal Manifestations

Gastrointestinal symptoms are common in patients with HIV infection, a recent study reported 72% of such patients developed gastrointestinal symptoms over a follow-up period of 6 months(31). Gastrointestinal microsporidiosis, typically presents with chronic watery diarrhoea with abdominal cramps. The prevalence of diarrhoea from microsporidial infection varies with geographical location and local diagnostic expertise and has been reported to be 7- 50% (20). Diarrhoea is non-bloody and intermittent with a frequency of 3-4 stools per day. Diarrhoea is progressive and worse in the mornings. Some patients report postprandial nausea, vomitting and abdominal pain. Fever is uncommon and weight loss is common. Most of the patients have evidence of carbohydrate and fat malabsorption. Electrolyte abnormalities particularly hypokalemia and hypomagnesemia are often present(27, 29). Majority of patients have evidence of advanced AIDS with CD4 counts typically <100/ mm^3 (20,22,27) although documented cases have been reported in patients with CD4 counts > 250/ mm^3 (32). Endoscopic appearance of small bowel mucosa is non-specific. Duodenal biopsies may reveal villous atrophy (20)

E. bieneusi is the most common microsporidium associated with diarrhoea in HIV infected patients.

structures measuring 1-2 x 1.5- 4 µm in size. Background debris stain faint green or blue. Correct diagnosis depends on finding the spores with distinctive species specific features like size, shape and staining pattern. The strong trichrome is useful on unconcentrated specimens as the usual concentration methods may lead to loss of spores (43).

Warthin -Starry Stain

Warthin-Starry may be useful for identifying microsporidial spores in staining tissue sections. It is not routinely used to stain stools and other fluid specimens because of high background staining.

Fluorescent Microscopy

Chemofluorescent agents are organic compounds that act as optical brighteners. They bind to chitin and cellulose and, upon excitation by short wavelength light, emit light in the visible spectrum. They have been used for rapid screening of stool specimens but lack specificity as degenerating bacteria and fungi also stain leading to difficulties in interpretation (46). These methods are not widely available and require a fluorescent microscope.

Immunofluorescence

Polyclonal and monoclonal antibodies have been raised against cultured E. hellem and E. cuniculi used in immunofluorescent staining to identify microsporidial spores (47). Immunofluorescent antibody staining is a better method of identifying microsporidia in tissue sections, but less sensitive than modified trichrome for detecting microsporidia in in stool specimens (48). However these methods are not yet available for general diagnostic use.

Polymerase Chain Reaction

Polymerase Chain Reaction (PCR)based methods are being developed to allow specific and reliable diagnosis of microsporidiosis. Gene encoding small subunits of ribosomal RNA followed by amplification using universal or species specific primers can be used to identify microsporidia species by restriction fragment length polymorphism analysis (49-51). Southern analysis can also be used in diagnosis and characterization studies. PCR primers have been used to diagnose disseminated microsporidiosis and differentiate different microsporidia from each other in clinical and cultured specimens (50). These methods are not widely available at present but when standardized and available, may be of great value in routine diagnosis of microsporidiosis in patient specimens.

Histopathology

. Histological diagnosis in gut and urinary tract specimens may be difficult with H & E staining and requires expertise. Specialized tissue staining with Gram stain, silver stains and PAS staining have been described but require expert interpretation and usually confirmation by electron microscopy.

Transmission Electron Microscopy

At present electron microscopy is the gold standard for diagnosis of microsporidiosis on tissue specimens. This is particularly useful in defining the microsporidial ultrastructure. This allows identification of distinctive features of spores as well as other stages of the parasite and easy differentiation of different species. Electron microscopy of urine and stool specimens is of low yield due to low concentration of spores. Debris and bacteria in stool specimens also make electron microscopy difficult. Methods for cleaning and concentration of fecal specimens before electron microscopy are being developed and may be useful (52).

Serology

Serological tests have traditionally been used to diagnose microsporidiosis in immunocompetent hosts. The use of serological tests in immunocompromised patients present many difficulties. Progressive immunodeficiency and inability to produce antibodies in later stages are complicated by inability to Considerable inter-species cross-reactivity is seen as well as cross reactivity between microsporidia and other parasites like cryptosporidium (53-55). Serological studies have shown that only 50% of HIV-infected patients with microsporidiosis may produce high levels of specific antibodies and over 50% of patients without HIV or microsporidial infection may also express significantly high levels of antibodies (53, 55). At present no reliable serological test is available for diagnosis of microsporidiosis in humans.

Diagnosis of Hepatobiliary Microsporidiosis

The laboratory findings in patients with biliary microsporidiosis typically reveal anicteric cholestasis with high alkaline phosphatase levels (4). The transaminases are less than twice normal and serum bilirubin rarely exceeds 2mg/dl (4, 11). The serum amylase may be elevated in about 10% of patients. The CD4 count is almost always less than 100 (40)

Abdominal ultrasonography is usually the first diagnostic modality in patients suspected to have AIDS cholangiopathy. Abnormalities are found on ultrasound in 75% cases with AIDS related cholangiopathy (4,11,39). Dilatation of the common bile duct is noted in 65% to 75% of cases and thickening of the common duct wall in 20% to 40% (3, 11, 39). Gall bladder wall thickening is noted in more than 50% cases but cholelithiasis is distinctly rare (11, 39). Benhamou et al studied endoscopic ultrasound findings in AIDS related cholangitis and reported that endoscopic ultrasound may be better than trans-abdominal ultrasound in defining dilatation and thickening of bile duct(39). Hepatobiliary scan (HIDA) may show ductal dilatation and hepatic parenchymal retention suggestive of AIDS related cholangitis and gall bladder may not be visualized if acalculous cholecystitis is present(50). ERCP and ultrasound are complimentary in the diagnosis of AIDS cholangiopathy(3) although ERCP is considered to be the gold standard(1). C T scan findings are similar to those of ultrasound although CT may be better in demonstrating the intrahepatic ductal dilatation and ultrasound better in demonstrating the common duct thickening and strictures(1).

The diagnosis of AIDS related cholangitis is based on a typical cholangiographic pattern in a patient with advanced AIDS, supported by identification of a pathogenic organism. Cello(4) has described four distinct cholangiographic patterns associated with AIDS cholangiopathy. The pattern of papillary stenosis, is characterized by dilatation of the common bile duct to > 8 mm with smooth tapering of the distal duct and occurs in 15- 20 % of patients. Sclerosing cholangitis is characterized by focal strictures and dilatations of the intrahepatic and extrahepatic ducts and occurs in about 20% of patients. Combined paillary stenosis and sclerosing cholangitis occurs in 50% cases. The pattern of long , 1-2 cm extrahepatic bile duct strictures occurs in 15% cases.

Pathogens identified in stools, bile or in biopsy specimens from papilla and duodenum may provide circumstantial evidence that the pathogen is the cause of cholangitis. However, a biopsy from the bile duct demonstrating a pathogen and associated inflammation is direct evidence that the pathogen is actually cause of the cholangitis. Aspiration of bile is essential particularly for demonstrating microsporidia(10). In patients with biliary cryptosporidiosis the organism is almost always present in the stool(11). It is essential to obtain biopsies from multiple sites to increase the diagnostic yield(11). In 75 % patients with AIDS related cholangiopathy pathogens are identified(8,39). Most of the cases are diagnosed on routine microscopy with appropriate stains, although advanced techniques and electron microscopy mav be required to confirm the diagnosis in some.

THERAPY

Therapeutic options for microsporidial infections are limited at present. The initial enthusiasm for metronidazole has subsided and only albendazole has shown some promise in open label and anecdotal studies. Albendazole is believed to interfere with tubulin polymerization and inhibit microtubule formation. Given in doses of 400 mg twice daily for 3-4 weeks, albendazole reduces diarrhea and promotes weight gain. Albendazole does not usually eradicate E. bieneusi infection and relapse occur after cessation of therapy(15, 28, 57, 58), although better results are seen in E. intestinalis infection with higher eradication rates and resolution of clinical symptoms(15,28). However relapses are also seen in E. intestinal infections after stopping treatment. Molina et al treated 5 patients with disseminated E. intestinalis infection with albendazole and noted rapid clinical improvement and clearance of the organism from stools of all 5 patients and urine of 3 patients(15). A multicenter, placebo controlled trial of albendazole therapy is near completion(23).

In one study, 12 patients who relapsed on albendazole therapy for microsporidial diarrhoea were reported to experience reduction in diarrhea and weight gain on thalidomide 100 mg nightly for 3 weeks(59). The mechanism of action thalidomide is believed to be down-regulation of the inflammatory cytokine TNF-a(20).

Atovaquone has been reported to improve symptoms in 8 patients with E. bieneusi infection without eradicating the organism. A double blind, placebo controlled trial is in progress evaluating this agent.(60). Nitazoxamide is currently being evaluated in microsporidial infection in a study in New York City(23).

Carr et al treated five HIV infected patients with chronic microsporidiosis and 3 patients with cryptosporidiosis with combination anti-retroviral therapy including a protease inhibitor. All patients had clinical response including two patients with biliary microsporidiosis(61). They believe that combination antiretroviral therapy can restore immunity to opportunistic organisms and may result in clinical and microbiological response.

Asmuth et al have advocated low fat, low residue diets coupled with antisecretory agents pending specific and effective anti-microsporidial therapy(34). Octreotide appears to be of some benefit in microsporidial diarrhea(62)

Treatment of AIDS Related Cholangitis

Medical therapy in AIDS related cholangitis has not been successful in most cases. Both gemicyclovir and foscarnet have not been successful in influencing the course of CMV related cholangitis(11,63) and paromomycin and azithromycin do not seem to be useful in biliary cryptosporidosis(40). Albendazole has been used with some success in disseminated infection including cholangitis with E. intestinalis(15). However the therapeutic effect seems to be transient. Ursodeoxycholic acid has been reported to improve cholestasis and pain in AIDS related cholangitis when patients failed to respond to sphincterotomy(61). Endoscopic sphincterotomy has been reported to significantly improve pain in AIDS related cholangitis in most of the published series(1,11,65) except one(40). Biliary stents have been used in patients with long strictures and proximal strictures but the experience is limited(1)

PROGNOSIS

Microsporidial infection is a manifestation of advanceed AIDS and usually associated with low CD-4 counts and poor survival(20). The prognosis of AIDS related cholangitis is particularly poor as it is a manifestation of advanced AIDS. The median survival is 7 months(63), although rarely prolonged survival to 32 months has been reported(12). The poor prognosis is not related to AIDS related cholangitris per se but reflects the severity of underlying immunodeficiency state.

References

1. Nash JA; Cohen SA. Gallbladder and biliary tract disease in AIDS. Gastroenterology Clinics of North America, 1997; 26(2):323-35.
2. Schneiderman DJ. Hepatobiliary Abnormalities of AIDS. Gastroenterol Clin North Am 1988; 17; 615
3. Urbain D, Jeanmart J, Lemone M, et al. Cholestasis in patients with the acquired immunodeficiency syndrome: comparison between ultrasonographic and cholangiographic findings. Am J Gastroenterol 1991; 86: 574.
4. Cello JP. Acquired immuno-deficiency syndrome chollangiopathy: spectrum of disease. Am J Med 1989; 86: 539.
5. Guarda LA, stein SA, Cleary KA, et al. Human cryptosporidiosis in the acquired immunodeficiency syndrome. Arh Pathol Lab Med 1983; 107: 562.
6. Pitlick S, Fainstein V, Rios A, et al. Cryptosporidial cholecystitis. N Eng J Med 1983; 308: 967.
7. Margulis SJ, Honig CL, Souve R, et al. Biliary tract obstruction in the acquired immunodeficiency syndrome. Ann Int Med 1986; 105: 207.
8. Farman J, Brunetti J, Baer JW, et al . AIDS-related cholangiopancreatographic changes. Abdominal Imaging, 1994 Sep-Oct, 19(5):417-22.
9. Blumberg RS, Kelsey P, Perrone T, et al. Cytomegalovirus and Cryptosporidium associated acalculous gangrenous cholecystitis. Am J Med 1984; 76: 1118.
10. Pol S, Romana C, Richard S, et al. Microsporidia infection in patients with human immunodeficiency virus and unexplained cholangitis. New Eng J Med 1993; 328: 95.
11. Bouche H, Housset C, Dumont JL. AIDS related cholangitis: diagnostic features and course in 15 patients. J Hepatol 1993; 17: 34.
12. Cello Jp, Chan MF. Long term follow up of endoscopic retrograde cholangiopancreatography and sphincterotomy for patients with acquired immunodeficiency syndrome and papillary stenosis. Am J Med 1995; 99: 600.
13. Orenstein JM, Tenner M, Cali A, et al. ·Microsporidian, previously undescribed in humans, infecting enterocytes and macrophages and associated with diarrhea in an AIDS patient. Hum Pathol 1992; 23: 722- 28.
14. Kotler DP; Orenstein JM. Prevalence of intestinal microsporidiosis in HIV-infected individualsreferred for gastroenterological evaluation. American J Gastroenterol, 1994; 89(11):1998-2002.
15. Molina JM; Oksenhendler E; Beauvais B; et al. Disseminated microsporidiosis due to Septata intestinalis in patients with AIDS: clinical features and response to albendazole therapy. Journal of Infectious Diseases, 1995; 171(1): 245-9.
16. Orenstein JM; Gaetz HP; Yachnis AT; et al. Disseminated microsporidiosis in AIDS: are any organs spared? [letter]. AIDS, 1997 Mar, 11(3):385-6.
17. Gunnarsson G, Hurlbut D, DeGirolami PC, et al. Federman M; Wanke C. Multiorgan microsporidiosis: report of five cases and review. Clin Infect Dis 1995; 21(1): 37- 44.
18. Human microsporidiosis: past and present, an overview of the Cleveland workshop. Journal of Eukaryotic Microbiology, 1994 Sep-Oct, 41(5):69S.
19. Wilson R, Harrington R, Stewart B, et al. Human immunodeficiency 1- associated necrotizing cholangitis caused by infection with Septata intestinalis. Gastroenterol 1995; 108: 247- 51.
20. Croft SL, Williams J, McGowan I. Intestinal microsporidiosis. Seminars in Gastrointestinal Disease 1997; 8(1): 45-55.
21. Canning EU. Microsporidia, in Kreier JP (ed): Parasitic Protozoa (2nd ed). San Diego, CA, Academic Press Inc, 1993, pp 299-370.
22. Conteas CN, Didier ES, Berlin OG. Workup of gastrointestinal microsporidiosis. Digestive Disease 1997; 15 (6): 330- 45.
23. Framm SR, Soave R. Agents of diarrhea. Med Clin North Am 1997; 81; 427- 47.
24. WeberR, Bryan Rt. Microsporidial infection in immunodeficient and immunocompetent patients. Clin infect Dis 1994; 19; 517.
25. Bryan RT. Microsporidiosis as an AIDS-related opportunistic infection. Clinical Infectious Diseases1995; 21 Suppl 1: S62-5.
26. Desportes I, LeCharpentier Y, Galian A, et al. Occurrence of a new microsporidian: Enterocytozoon bieneusi n.g., n.sp., in the enterocyte of a human patient with AIDS. J Protozool 1985; 32: 250.
27. Weber R, Bryan RT, Schwartz DA, et al. Human microsporidial infections. Clin Microbiol Rev 1994; 7- 426.
28. Dore GJ; Marriott DJ; Hing MC; et. Disseminated microsporidiosis due to Septata intestinalis in nine patients infected with the human immunodeficiency virus: response to therapy with albendazole. Clinical Infectious Diseases 1995; 21(1): 70-6.
29. Cali A, Kotler DP, Orenstein JM. Septata intestinalis, N.G, N.Sp, an intestinal microsporidian associated with chronic diarrhea and dissemination in AIDS patients. J Eukaryot Microbiol 1993; 40: 101- 12.
30. Beauvais B, Sarfati C, molina JM, et al. Comparative evaluation of five diagnostic methods for demonstrating microsporidia in stool and intestinal biopsy specimens. Ann Trop Med Parasitol 1993; 87: 99- 102.
31. May GR, Gill MJ, Church DL, et al. Gastrointestinal symptoms in ambulatory HIV- infected patients. Dig Dis Sci 1993; 38: 1388- 94.
32. Sowerby TN, Conteas CN, Berlin OGW, et al. Microsporidiosis in patients with relatively preserved CD4 counts. AIDS 1995; 9: 975.
33. Orenstein Jm, Chiang J, Steinberg W, et al. Intestinal microsporidiosis as a cause of diarrhea in human immunodeficiency virus-infected patients: a report of 20 cases. Hum Pathol 1990; 21: 475-81.
34. Asmuth DM, DeGirolami PC, Federman M, et al. Clinical features of microsporidiosis in patients with AIDS. Clinical Infectious Diseases 1994; 18(5): 819-25.
35. Beaugerie L, Teilhac MF, Deloul AM, et al. Cholangiopathy associated with microsporidia infection of the common bile duct mucosa in a patient with HIV infection. Ann Int Med 1992; 117: 401-02.

36. Weber R, Kuster H, Keller R, et al. Pulmonary and intestinal microspridiosis in apatient with the acquired immunodeficiency syndrome. Am Rev Resp Dis 1992; 146: 1603- 5.
37. Orenstein JM, Dieterich DT, Kotler DP, et al. Systemic dissemination by a newly recognized intestinal microsporidia species in AIDS. AIDS 1992; 6: 1143-50.
38. Pitlick S, Fainstein V, Garza D, et al. Human cryptosporidiosis: spectrum of disease: report of six cases and review of the literature. Arch Intern Med 1983; 143: 2269.
39. Benhamou Y, Caumes E, Gerosa Y, et al. AIDS- related cholangiopathy: critical analysis of a prospective series of 26 patients. Dig Dis Sci 1993; 38: 1113.
40. Vakil NB, Schwartz FN, Buggy BP, et al. Biliary cryptosporidiosis in HIV-infected people after the waterborne outbreak of cryptosporidiosis in Milwaukee. N Eng J Med 1996; 334: 19.
41. Hollister WS, Canning EU, Colbourn NI, et al. Encephalitozoon cuniculi isolated from the urine of an AIDS patient, which differs from canine or murine isolates. J Eukaryot Microbiol 1995; 42: 367- 72.
42. VanGool T, Hollister WS, Eeftinck Schattenkerk JKM, et al. Diagnosis of Enterocytozoon bieneusi microsporidiosis in AIDS patients by recovery of spores from faeces. Lancet 1990; 336; 697-98.
43. Weber R, Bryan RT, Owen RL, et al. Improved light-microscopical detection of microsporidial spores in stool and duodenal aspirates. N Eng J Med 1992; 326: 161-66.
44. Ryan NJ, Sutherland G, Coughlan K, et al. A new trichrome-blue stain for detection of microsporidial species in urine , stool and nasopharyngeal specimens. J Clin Microbiol 1993; 431: 3264- 69.
45. Kokoskin E, Gyorkos T, Camus A, et al. Modified technique for efficient detection of microsporidia. J Clin Microbiol 1994; 32: 1074-75.
46. VanGool T, Snijders F, Reiss P, et al. Diagnosis of intestinal and disseminated microsporidial infections in patients with HIV by anew rapid fluorescence technique. J Clin Pathol 1993; 46: 694-9.
47. Aldras AM, Orenstein JM, Kotler DP, et al. Detection of microsporidia by indirect immunofluorescence antibody test using polyclonal and monoclonal antibodies. J Clin Microbiol 19994; 32: 608- 12.
48. Didier ES, Orenstein JM, Aldras AM, et al. Comparison of three staining methods for detecting microsporidia in fluids. J Clin Microbiol 1995; 33: 3138- 45.
49. Schuitema ARJ, Hartskeeerl RA, VanGool T, et al. Application of the polymerase chain reaction for the diagnosis of microsporidiosis. AIDS 1993; 7(Suppl 3): 57-61.
50. Fedorko DP, Nelson NA, Cartwright CP, et al. Identification of microsporidia in stool specimens by using PCR and restriction endonucleases. J Clin Microbiol 1995; 33: 1739- 41.
51. Baker MD, Vossbrinck CR, Didier ES, et al. Small subunit ribosomal DNA phylogeny of various microsporidia with emphasis on AIDS related forms. J Eukaryot Microbiol 1995; 42: 564- 70.
52. Corcoran GD, Tovey DG, Moody AH, et al. Detection and identification of gastrointestinal microsporidia using non-invasive techniques. J Clin Pathol 1995; 48: 725-27.
53. Ombrouck c, Romestand B, Da Costa JMC, et al. Use of cross reactive antigens of the microsporidian Glugea atherinae for the possible detection of Enterocytozoon bieneusi by Western blot Am J Trop Med Hyg 1995; 52: 89- 93.
54. Hollister WS, Canning EU, Willcox A. Evidence for widespread occurrence of antibodies to Encephalitozoon cuniculi (microspora) in man provided by ELISA and other serological tests. Parasitology 1991; 102: 33- 43.
55. Didier ES, Kotler DP, Dieterich DT, et al. Serological studies in human microsporidiosis. AIDS 1993; 7(suppl) : 8- 11.
56. Cacciarelli AG, Marano BZ, Zurelti AR. Gall bladder disease in the HIV population. Gastroenterology 1995; 108: A 407.
57. Dieterich DT, Lew EA, Kotler DP, et al. Treatment with albendazole for intestinal disease due to Enterocytozoon bieneusi in patients with AIDS. J Infect Dis 1994; 169: 78-83.
58. Blanshard C, Ellis DS, Tovey DG, et al. Treatment of intestinal microsporidiosis with albendazole in patients with AIDS. AIDS 1992; 6: 311- 13.
59. Sharpstone D, Rowbottom A, Francis N, et al. Thalidomide: a novel therapy for microsporidiosis. Gastroenterology 1997; 112(6): 1823-9.
60. Anwar-Bruni DM, Hogan SE, Schwartz DA, et al. Atovaquone is effective treatment for the symptoms of gastrointestinal microsporidiosis in HIV-1-infected patients. AIDS 1996; 10(6): 619-23.
61. Carr A, Marriott D, Field A, et al. Treatment of HIV-1-associated microsporidiosis and cryptosporidiosis with combination antiretroviral therapy. Lancet 1998; 351: 256-61.
62. Simon DM, Cello JP, Valenzuela J, et al. Multicenter trial of octreotide in patients with refractory acquired immunodeficiency syndrome-associated diarrhea. Gastroenterology 1995; 108: 1753-60.
63. Forbes A, Blaanshard C, Gazzard B. Natural history of AIDS-related sclerosing cholangitis: a study of 20 cases. Gut 1993; 34: 116.
64. Chan MF, Koch J, Cello JP. Ursodeoxycholic acid for symptomatic AIDS-associated cholangiopathy. Gastrointest Endosc 1994; 40: A 108.
65. Lynche KD, Savides TJ, Weiner KJ. AIDS cholangiopathy: the role of therapeutic ERCP. Gastrointest Endosc 1994; 40: a 117.

TEA AND HEALTH

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INTRODUCTION

Tea is a popular drink, enjoyed everyday by millions of people around the world. Drinking tea is a safe, inexpensive and pleasurable experience. Tea also provides a natural source of compounds that protect against a wide array of disease.

Tea is made from brewing leaves of the plant camellia sinensis in hot water. Three different kinds of brew are popular green tea, oolong tea and black tea. Uptill now it was believed that only green tea provided health benefits. However, recent studies have dispelled this myth. All three teas are a rich source of antioxidants called flavonoids, vitamins and fluoride.

TEA AND CARDIOVASCULAR DISEASE

There is growing evidence suggesting that flavonol / flavonoid or tea consumption is linked to protection from ischemic heart disease and stroke(1,2). Conclusions from a recent meta analysis suggest that drinking 3 cups of tea a day, reduces risk of myocardial infarction by 11%.

Mechanisms by which tea may confer its cardiovascular properties include prevention of LDL oxidation(4), reduction in blood lipids(5), anti inflammatory action(6), improvement in blood vessel function(7) and inhibition of platelet aggregation(8). Although some epidemiological studies seem to indicate that tea can reduce serum cholesterol(9), most studies show no effect in reduction of cholesterol or blood pressure.

As antioxidant activity from tea has been demonstrated in vivo(10), it may be that tea exerts its cardio protective properties in other ways such as improvement in endothelial function, platelet aggregation or ways that have yet to be investigated. Although scientific evidence for the mechanisms by which tea exerts its positive health effects is growing, it is not yet conclusive and represents promising areas for future research in human trials.

TEA AND CANCER

Cancer is eventually a disorder of cellular genetic material. Damage to the DNA within the cells may be as a result of environmental factors such as radiation, chemical carcinogens and some viruses.

More and more research is now indicating that diet plays a crucial role in the prevention of cancer. Specific substances, such as phenolic compounds found in plants can enhance general healthy eating recommendations. Flavonoid is one such phenolic compound present in tea.

Flavonoid are powerful antioxidants and have been identified as potential cancer preventative components(11). Tea has been shown to inhibit tumorigenesis at the initiation, promotion and progression stages of cancer(12). Possible mechanisms of action include antioxidant activity, ability to inhibit nitrosamine reactions(13), modulation of carcinogen metabolising engymes(14) trappening of ultimate carcinogens(15), ability to inhibit all proliferation(15), modulation of gut microflora associated with colon cancer(16), antimicrobial action associated with helicobacter pylori and gastric cancer(17).

Although there are conflicting reports on association between tea drinking and stomach cancers, a recent one suggests that tea drinkers had a 48% reduced risk of developing stomach cancer and a 51% lower risk of developing chronic gastritis versus non drinkers(18)

Flavonoids have also been associated with a reduced risk of lung cancer. A case control study has reported 50% reduction in lung cancer risk when consuming 1 cup of black tea a day(19). A recent study in Japan shows that regular consumption of green tea, more than 3 cups a day might be protective against breast cancer in early stages(18).

TEA AND HYDRATION

It was widely believed in the part that tea should be avoided as a fluid replacement due to the diuretics action of caffeine present in tea.

An extensive review carried out by prof. Ron Haughan and Jane Griffen concluded that there is no evidence for the assumption that all caffeine containg drinks should be avoided in situation where fluid balance is precarious(20). It has been found that tea does not have a diuretic effect due to caffeine under the amount of tea consumed at one sitting contains more than 250-300 mg of caffeine, equivalent to 5-6 cups of tea(21).

In fact, due to the volume of fluid that is drunk while enjoying a cup of tea, it can contribute towards the daily

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TABLE 1

NUTRIENT	Amount provided by 3 cups of tea alone	Percentage of daily recommended nutrient intake provided by tea alone	Amount provided by the semi-slummed with present in 3 cups of tea	% of daily recommended nutrient intake provided by milk alone in 3 cups of tea	Total amount provided by 3 cups of tea with demi skimmed milk
MINERALS			108 mg	15%	108 mg
CALCIUM	Trace		0.36mg	5% - females	0.36 mg
				4% - males	
ZINC	Trace	3%	135mg	4%	232 mg
Potassium	97mg				0.8 mg
Manganese	0.8mg				
VITAMINS			30mcg	5% - females	36 mg
				4% males	
Thiamine	Trace	5% Females	162 mcg		219 mcg
		4% Males			
Riboflavine	56 mcg		54 mcg	15% females	54 mcg
				13% males	
Pyridoxine	Trace		0.9mg		5.4 mcg
Folic Acid	Trace	4% Females	0.3 mg	3%	0.66 mg
		3% Males			
Niacin	0.57 mg		0.36 mg	1% Females	0.3 mg
				1% Males	0.36 mcg
Pantothenate	Trace				
Vit B-12	Nil			24%	

1 cup = 190 ml, 1 cup of tea having 30 ml milk. In addition to the above nutrients, tea provides 70% of our daily fluoride intake.

recommended fluid intake of 1.5 to 2 litres.

TEA AND DENTAL HEALTH

Epidemiological studies have reported that some populations who drink tea on a regular basis have a reduced number of carious teeth22 various mechanisms have been proposed...

(a) Fluoride – Tea is a very rich source of fluoride, 1 cup of tea contains between 0.3 mg – 0.5 mg of fluoride. A recent study has demonstrated that after rinsing with tea, 34% of the fluoride was retained in the oral cavity and some of this showed a strong binding ability to enamel particles and potentially helping to prevent dental decay. Some concern has been raised over excessive fluoride intake & fluorosis in children, However Dental profession is almost totally in favour of water fluoridation & any concerns have been carefully considered & discounted by the Public Health Alliance & the British Fluoridation Society(1995).

(b) Tannins – It has been reported that the tannins in tea can inhibit salivary amylase thereby reducing the carcinogenic potential of starch containing foods(23) A number of studies have also demonstrated that tannic acid inhibits the growth of s. mutans bacteria(24), a major factor in the build up of dental plaque.

(c) Acid erosion – Components of tea – tannin, catechin, caffeine & tocophenol have been shown to be effective in increasing acid resistance of tooth enamel(25). Their effects increased dramatically when used in combination with fluoride.

(d) Flavonoids – Green tea, and specific flavonoids, mainly catechins, have inhibitory effects on growth of carcinogenic bacteria by inhibiting adherence and growth of plaque bacteria.

TEA DRINKING & IRON ABSORPTION

Tea drinking mainly influences the absorption of non haem iron as haem iron is relatively unaffected by tea.

Few studies have attempted to quantify the effect of tea on non haem absorption and have concluded that tea does have an inhibiting effect on iron absorption, however it may not reflect the role of tea when consumed as a part of a complex, real diet(26).

Short-term studies indicate that there may be a negative association between tea drinking and iron status. However, they do not necessarily show cause and effect i.e. the negative iron status is due to drinking tea. Instead it shows that a group who had a low iron status also had a higher intake of tea.

Tea consumption will not result in iron deficiency for healthy individuals who are consuming a varied and balanced diet. Hence iron is present in good amounts in meat, offal and is readily absorbed, unaffected by tea drinking. Non haem iron found in cereals, vegetables, dried fruits, legumes and nuts is less well absorbed and its absorption is influenced by a wide variety of dietary factors which include enhances (vit C, animal tissue) and inhibitions (phytate and tea). Hence, those having a poor iron status or there at risk of iron deficiency, should avoid drinking tea with meals.

Otherwise moderate tea drinking (3-4 cups) is unlikely to have any adverse effect on iron status, in fact it can actually bring about other health benefits.

NUTRITIONAL VALUE OF TEA

Tea alone has no calories; however adding milk can provide a no. of vitamins and minerals. Table 1 depicts the nutrients present in 3 cups of tea with semi skimmed milk, as well as their contribution to recommended daily intakes(27, 28).

CONCLUSION

Studies have proved that tea flavonoids demonstrate antioxidant activity. Though not a replacement for fruits and vegetables, the antioxidant activity of tea is comparable.

As well as contributing to fluid and antioxidant intake, drinking 3-4 cups of tea a day with milk can provide certain vitamins and minerals, thereby helping to support overall health and well being.

Its protective effect against several diseases like cardiovascular diseases, cancer, dental caries and Bacterial infections have been shown by various epidemiological studies. Thus, tea seems to fit the description of an ideal component of a healthy dietary habit that can be used to promote human health. It is yet a promising area of research for future human studies.

REFERENCES

1. Stenevold 1, et al. Tea consumption relationship to cholesterol. BP & coronary diseases Preventive Med., 1992 21:546,.
2. Hertey MCL, et al. Dietary antioxidant flavonoids & risk of coronery heart disease: the zutphen olderly study: Lancet: 1993, 155 : 381.
3. Peters U, et al. Does tea effects cardiovascular disease: meta analysis Am J. Epidemiology: 2001 154; 6:459-501
4. Vinson JA et al. Plant flavonoids, especially tea flavonol, are powerful antioxidants using as in vitro oxidation model for heart disease, J. Agnic, Food Chem, 1995; 43 (11): 2800 – 2802.
5. Yang TTC, et al, Hypocholesterolemic effects of Chinese tea, Pharmacology, 1997; 35:505-512.
6. Tijbiug LMB, Tea flavonoids & CV diseases: A review, Food. Sci. Nut. 1997,37; 771.85
7. Duffy SJ et al short and long-term black tea consumption reverses endothelial dysfunction in patients with coronary artery disease 2001. Circulation 10,104,2,151.
8. Hodgsen JM, et al effects of regular ingestion of black tea on haemostasis and cell adhesion molecules in humans. Cur. J. Clin Nuth; 2001; 55(10) : 881-6.
9. Gmai k et al has sectioned study of effects of drinking green tea on CV and lives diseases. BMJ, 1998:310; 693-6.
10. Serafini M, et al In vivo oxidant effects of green and black tea in man Cur. J. Clin Nutr; 1996:50; 28-32.
11. Lynn looks BD et al chemo preventive effects of tea extracts. Nut. Cancer, 1999; 35:80-86.
12. Katiyar SK et al, Tea in chemo prevention of cancer: epidemiological and experimental studies (Review) In, J . Oncology, 19936:5; 425-38.
13. Wang ZY et al (Inhibition of N-Nitrosodiethylamine and 4-methylnitrosamine 1 butanone induced tumorigenesis in A/J mice by Green and black tea cancer Res. 1992; 52:1943-7.
14. Jakun J et al why drinking tea could prevent cancer Nature; 1997:387;561.
15. Yang CS et al. Tea and cancer J. Naff. Cancer Inst. 1993 480; 1038-49.
16. Yamamato T, et al. Chemistry and applications of green tea CRC press LLC: 1997, Boca Rotan, USA.
17. Diker KS, et al. The bactericidal activity of tea against Helicobacter pylori. Lett Appl. Microbio. 1994:19; 299-300.
18. Goldbohm RA et al. Intake of flavonoids and cancer risk: a prospective cohort study In:Cost 916. 1990, Polyphenols in food. 159-66.
19. Hendilaharsee M et al. Consumption of tea and coffee and the risk of lung cancer in cigarette smoking men: a case control study lung cancer. 1998 19, 101-7.
20. Ron Maughan and Jane Griffen. Tea drinking and fluid balance: A review. 2001. Brooke Bond Tea Company.
21. Martof. M.T. And Knox D.K. The effect of nanthines on fluid balance clinical. Nursing Research, 1997 .6: 186-196
22. Cao J et al. Observation of caries incidence among tea drinking population. J. Dental Health. 1987; 31:86-9.
23. Simpson A. et al. The bio availability of fluoride from black tea J. Dentistry: 2001, 29:15-21.
24. Itoh T. Inhibition of Carcinogenic factor of streptococci mutants by tannic acid. Nihon Univ. J. Oral Sci, 1991:17; 45-126.
25. Yu 4 et al. effects of several tea components on acid resistance of human tooth enamel. J. Dent. 1995:23; 101-105.
26. Gibson SA. Iorn intake and iron status of pre school children: Association with breakfast cereals vitamin C and meat. Public Health Nutrition 1999. 2:521-8.
27. Holland B, Welch AA et. Al Mc Cance and Widdowson's the composition of Foods, 5th Edition: 1991, Royal Society of Chemistry, Cambridge.
28. Doh(1991) Dietary reference values for food energy and nutrients for UK. Report of the panel.

CANCER CHEMOPREVENTION THROUGH NATURAL ANTIMUTAGENIC AGENTS

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Mutations in somatic cells are not only involved in the carcinogenesis but do also play a role in the pathogenesis of other chronic degenerative diseases such as atherosclerosis and other heart diseases[1]. Prevention of cancer and other such mutation-related diseases can be pursued both by avoiding exposures to recognized mutagens/carcinogens and by favouring the intake of protective factors which fortify physiological defense mechanisms. The latter approach referred to as Chemoprevention involves dietary or pharmacological intervention in the host organism[2].

It has been suggested that the use of antimutagens and anticarcinogens in everyday life will be the most effective procedure for preventing human cancer and genetic disorders[3]. To date more than 500 chemicals have been assayed as potential cancer chemopreventive agents, with representatives from at least 25 different classes showing protective effects[4]. However in recent years, there is increasing awareness that certain naturally occurring substances in plants and other natural sources provide potential protection against environmental mutagens/ carcinogens as well as endogenous mutagens[3]. Similarly, overwhelming evidence from several epidemiological studies has indicated that diverse dietary components are closely associated with reduced cancer risk by virtue of their antimutagenic properties[5].

Mechanisms of antimutagenesis:

Some of the mechanisms of antimutagenesis and anticarcinogenesis are listed as under: [3]

1. Bioantimutagens:
   These are equivalent to the "true" antimutagens. They act on the repair and replication processes of the mutagen-damaged DNA, resulting in a decline in mutation frequency.
2. Desmutagens:
   These must be considered only as apparent antimutagens. They indirectly inactivate the mutagens either fully or partially.
3. Chemical or enzymatic inactivators:
   Many mutagens, which are chemically reactive, acting not only on DNA but also on proteins and enzymes, may be directly inactivated by a range of different chemicals. Antimutagenic and anticarcinogenic properties have been associated with both inhibitors and inducers of cytochrome P450 enzymes such as indole-3-carbinol. Inducers of phase II enzymes such as glutathione transferase tend to inhibit a wide range of target carcinogens e.g., isothiocyanates such as benzyl isothiocyanate and antioxidants such as 2, 3-tert-butyl-4-hydroxyanisole (BHA).
4. Prevention of formation of active species:
   Many genotoxic carcinogens require metabolic activation to an electrophilic form that can react with DNA. Although these processes commonly occur in the liver, there is increasing evidence for metabolic activation by other tissues also, especially of the gastrointestinal tract. N-nitroso compounds are often formed in the stomach through a reaction from nitrite and secondary or tertiary amines.
5. Scavenging:
   A number of desmutagens are able to scavenge dietary mutagens (activated or non-activated) through binding or adsorption. In general, the mutagen remains intact during this process, but is unable to react with DNA. Chlorophyllin and some dietary fibers appear to act in this way.
6. Antioxidants and free radical scavengers:
   A wide range of chemopreventive agents has anti-oxidant or free-radical scavenging activity, e.g., carotenoids, retinoids and flavonoids. Once formed, free radicals, having a short half-life e.g., OH radical (half life = 10^{-9} sec), can be removed or inactivated.

Natural Products as Antimutagens:

Naturally occurring substances of plant origin and dietary components that have been widely studied for their antimutagenic activity include carotenoids, flavonoids, tea-polyphenols, vitamins, curcuminoids, tannins, coumarins, chlorophyllin, porphyrins, alkylresorcinols from cereal grains etc.

Vitamins

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Vitamin C and E have been shown to be anticlastogenic against doxorubicin induced chromosomal aberrations[6]; Vitamin C,E and A against. Ethyl-methane-sulphonate (EMS)-induced 6TG-resistant mutations in Chinese hamster V79 cells[7]. Vitamin A and E were found to be antimutagenic towards methylazoxymethanol (MAM)-induced mutagenesis in S. typhimurium strain TA100 [8].

Flavonoids:

Flavonoids present yet another class of antimutagens and anticlastogens with a high potential. Distinct structure – activity relationships were detected when 56 flavonoids, 32 coumarins, 5 naphthoquinones and 12 anthraquinones were tested for their antimutagenic potencies, with respect to mutagenesis induced by 2-nitrofluoro 3-nitro fluoranthene and 1-nitropyrine in S. Typhimurium TA 98. Among flavonoids, all flavones and many flavonoids with phenolic hydroxyl group like leuteolin, kaempherol etc. exerted antimutagenicity; chalcones and dihydrochalcones were potent antimutagens. Among compounds structurally related to anthraquinones, anthrone, acridone and xanthone exerted antimutagenicity; anthrone being the most potent one. All naphtho-quinones were potent antimutagens. Plumbagin and 2-methyl –5 hydroxynaphthoquinone showed exceptional antimutagenicity[9]. Wall et al. (1988)[10] observed non-toxicity and high activity of several coumarins, including psoralen, from *Psoralea coryliforia* and imperatorin and osthol from *Selinum monniere*, in the inhibition of mutagenicity of benzo(a)pyrene.

Citrus flavonoid tangeretin was found to be antimutagenic against the mutagens, benzo(a)pyrene, 2-amino fluorene, quercetin, and nitro-quinoline N-oxide, whileas nobiletin acted as an antimutagen against benzo(a)pyrene[11]. Manikumar et al., (1989)[12] isolated two new iso-flavones, fremontin and fremontone from roots of *Psorothamnus fremontin* and found both of them to be highly active in the inhibition of mutagenicity of ethyl methane sulfonate (EMS) at all concentrations tested. Antimutagenic effect of hispidulin and hortensin, the flavonoids from *Millingtonia hortensis* was seen[13] when tested against 2-aminoanthracene, aflatoxin B1 (in TA 98) and dimethyl nitrosamine (in TA 100).

Carotenoids

Several studies on carotenoids have shown that they affect activation of promutagens. The water-insoluble residues of some carotenoid-rich fruits and vegetables, such as apricots, oranges, brussels, sprouts, carrots, yellow-red peppers and tomatoes when sequentially extracted with several solvents[14] and tested for inhibition of mutagenicities induced by aflatoxin B_1, benzo(a)pyrine (BaP), imidazoquinoline and cyclophosphamide (CP) in histidine-deficient strains of S. typhimurium, number of BaP or CP-induced micronuclei in PCEs in bone-marrow of mice was reduced significantly by the carotenoids lycopene, canthaxanthin, lutein and beta-cryptoxanthin. Antimutagenicity of carotenoids extracted from five different types of green peppers *(Capsicum spp.)* has been reported[15] on S.typhimurium tester strain YG1024, against the mutagenicity of some nitroarenes. In 1997, Gonz'alez et al.[16] used the S.typhimurium tester strain YG1024 in the plate-incorporation test to examine the antimutagenicity of xanthophylls extracted from Aztec Marigold *(Tagetes erecta)* on 1-nitropyrene mutagenicity.

Phenolic Compounds

Certain phenolic/polyphenolic compounds present in various herbal teas have been found to be effective inhibitors of chemical mutagens and/or carcinogens. Polyphenols extracted from Chinese green tea (GTP) and its epicatechin derivatives exhibited antimutagenic effects in several test systems[17]. They were found to interact with hepatic cytochrome P-450 and inhibited the P450-dependent mixed function oxidase enzymes in skin and liver. Phenolics present in turmeric and clove, namely curcumin and eugenol respectively were found to inhibit the mutagenicity produced by direct acting mutagens such as N-methyl-N-nitro-N-nitrosoguanidine and sodium azide using S. typhimurium strains TA100 and TA1535 [18].Sukumaran (1995)[19] has reported that eugenol inhibited tobacco-induced mutagenesis in Ames test.

Herbal Teas

Tea is the most popular beverage consumed by over two thirds of the world's population. Tea is processed differently in different parts of the world to give green (20%), black (78%) or oolong tea (2%). Green tea is consumed mostly in Japan and China. The chemical components of green and black tea are polyphenols, which include Epicatechin, Epigallocatechin, Epigallin and Tocopherols. The antimutagenic activity of tea extracts and polyphenols including Epicatechin and Epigallocatechin has been demonstrated in microbial system (Ames test), mammalian cell systems and in-vivo animal tests[20].Antimutagenic effects of green tea against smoke-induced mutations in humans were investigated by Lee et al., 1997[21]. It was found that green tea can block the cigarette-induced increase in sister-chromatid exchange frequency.

Essential oil of Cymbopogon citratus Stapf., that is used as a constituent of "Lemongrass Tea" in central and southern parts of India has been shown to possess antimutagenic activity against lead nitrate and cyclophosphamide-induced micronuclei and chromosomal aberrations in-vivo in Swiss albino mice[22].

turmeric, mustard, green leafy and allium species of vegetables[49]. A human intervention study with vegetable products has been performed in twenty-three healthy human volunteers in order to observe whether consumption of vegetables containing different carotenoids could protect against DNA damage and oxidative DNA damage. It was seen that the supplementation of diet with tomato, carrot or spinach products resulted in a significant decrease in endogenous levels of strand breaks in lymphocyte DNA [50].

The anticlastogenic properties of two dietary supplements, garlic and mustard oil, were observed against the clastogenic activity of sodium arsenite [51]. Casein showed a strong antimutagenic activity in-vivo and ex-vivo in the DNA-repair host-mediated assay and liquid suspension assay, respectively [52]. Yogurt (milk fermented by a mixture of bacteria) was reported to be antimutagenic independently by Bakalinsky et al. (1996)[53]. Groves and Bala, 1993[54] reported antimutagenic effects of guava (*Psidium guajava*) in S.typhimurium. The mechanism of antimutagenic effects of mushrooms (Craterellus cornucopioides) was found to be by direct chemical interaction with the mutagens aflatoxin B_1, benzo(a)pyrene and acridine, and/or inhibition of the activation process in the case of promutagens[55]. A chloroform extract of mint showing no mutagenic activity in the Ames assay effectively inhibited the mutagenicity of carcinogens/mutagens like BaP, AFB_1, MMS and extract of white Shamma (a complex mixture of powdered tobacco, slaked lime, ash, oils, spices and other additives[56]. Asafoetida and turmeric extracts have been found to inhibit microsomal activation dependent mutagenicity of 2-acetamido fluorene. Similar results were also obtained using curcumin and eugenol which are phenolics present in turmeric and clove respectively[18]. Separation by semi-preparative HPLC of the n-hexane extracts of carrots, tomatoes and oranges indicated that the antimutagenicity was mainly associated with the fractions of the hydrocarbon carotenoids (alpha- , beta-carotene, lycopene), the xanthophylls (beta-cryptoxanthin, lutein), and also the carotenolesters (oranges)[14].

Some Interesting Observations on Antimutagens:

Apart from these facts, some interesting observations have been made by some workers during their studies on antimutagens. Segaran and Shankel (1997)[57] studied the effects of known antimutagens, namely polyamines and related compounds on the development of drug/antibiotic resistance in a variety of strains. They found that polyamines produce strong antimutagenic effects against EMS and MMS-induced antibiotic resistance. McCarty (1997)[58] proposed that natural antimutagenic agents may prolong efficacy of human immunodeficiency virus therapy which is otherwise affected by the tendency of transfected human immunodeficiency virus therapy to mutate to drug-resistant forms. Therefore safe intakes of selenium, green-tea polyphenols and cruciferous vegetables were expected to prolong the efficacy of drug therapy in subjects infected with the human immuno-deficiency virus.

It is therefore concluded that increased consumption of green leafy vegetables, carrots, yellow and red fruits, whole grain cereals, nuts, beverages and green tea etc shall impart necessary protection against the genotoxic effects of mutagens/carcinogens present in environment, food, drugs, cosmetics, industrial waste etc and thereby help in prevention of cancer and other degenerative diseases like atherosclerosis, diabetes mellitus, ischaemic heart disease, rheumatoid arthritis, neurological disorders etc.

References:

01. DeFlora, Izzotti A,Randerath K,Randerath E, Lertas J. DNA adducts and chronic degenerative diseases-Pathogenic relevance and implications in preventive medicine. Mut Res 1996: 366 : 197- 238
02. DeFlora S, Izzotti A, Benniceili C.Mechanisms of antimutagenesis and anticarcinogenesis, Role in primary protection, in. G. Bronezetti, H. Hayatsu, S. DeFlora, M.D. Waters and D. M. Shankel (Eds.). Antimutagenesis and anticarcinogenesis. Mechanisms III, Plenum, New York,1992.
03. Ferguson LR. Antimutagens as cancer chemopreventive agents in the diet. Mut Res 1994: 307 : 395-410
04. Boone CW, Kelloff GJ, Malone W. Identification of candidate cancer chemopreventive agents and their evaluation in animal models and human clinical trials; a review. Cancer Res 1990: 50 : 2-9
05 DeMarini DM.Dietary interventions of Human Carcinogensis. Mut Res1998:400 (1-2):457-65
06. Antunes LM,Takahashi CS.Effects of high doses of vitamin C and E against doxorubicin – induced chromosomal damage in wistar rat bone marrow cells. Mut Res 1998: 419 (1-3) : 137-43
07. Kuroda Y. Antimutagenic activity of vitamins in cultured mammalian cells. Basic Life Sci 1990: 52: 233-56
08. Tavan E, Maziere S, Narbonne JF, Cassand P. Effects of vitamins A and E on methylazoxymethanol-induced mutagenesis in S. typhimurium strain TA100. Mut Res 1997: 377 (2) : 231-7
09. Edenharder R,Tang X.Inhibition of the mutagenicity of 2-nitrofluorene, 3-nitrofluoranthene and 1-nitropyrene by flavonoids, coumarin, quinones and other phenolic compounds. Food chem Toxicol 1997: 35 (3-4) : 357- 72
10. Wall ME,Wani MC,Manikumar G, Hughes TJ, Taylor H, Warner J. Plant antimutagenic agents, 3. Coumarins. J Nat Prod 1988: 51 (6) : 1148-52
11. Calomme M, Pieters L,Vlietinck A,Vandenberghe D.Inhibition of bacterial mutagenesis by citrus flavonoids. Planta Med 1996:62 (3): 222-6
12. Manikumar, G., K. Gaetano, M.C. Wani, H. Taylor, T.J. Huges and M.E. Wall (1989) Plant antimutagenic agents, 5. Isolation and structure of two new isoflavones, fremontin and fremontone from Psorothamnus fremontii. J. Nat. Prod., 52 (4) : 769-73
13. Chulasiri M, Bunyapraphatsara N, Moongkarndi P. Mutagenicity and antimutagenicity of hispidulin and hortensin, the flavonoids from Millingtonia hortensis L. Environ Mol Mutagen 1992:204 (4) : 307-12

14. Rauscher R, Edenharder R, Platt KL.In-vitro antimutagenic and in-vivo anticlastogenic effects of carotenoids and solvent extracts from fruit vegetables rich in carotenoids. Mut Res 1998: 413 (2): 129-42
15. Gonzalez ME, Hernandez Q, Loarca G. Antimutagenic activity of carotenoids in green peppers against some nitroarenes. Mut Res 1998: 416 (1-2) : 9-11
16. Gonzalez ME,Loarca G,Ramos GM. Antimutagenicity of xanthphylls present in Aztec Marigold (Tagetes erecta) against 1-nitropyrene. Mut. Res. 1997:389 (2-3):219-26
17. Mukhtar H, Wang ZY, Katiyar SK,Agarwal R.Tea components: antimutagenic and anticarcinogenic effects. Prev Med 1992:21 (3) : 351-60
18. Soudamini KK, Unnikrishnan MC, Sukumaran K, Kuttan R. Mutagenicity and anti-mutagenicity of selected spices. Ind J Physiol Pharmacol 1995: 39 (4) : 347-53
19. Sukumaran K,Kuttan R. Inhibition of Tobacco-induced mutagenesis by eugenol and plant extracts. Mut Res 1995: 343 (1) : 25-30
20. Kuroda Y,Hara Y.Antimutagenic and anticarcinogenic activity of tea polyphenols. Mut Res 1999: 436 (1) : 69-97
21. Lee IP, Kim YH, Kang MH, Roberts C, Roh JK.Chemopreventive effect of green tea (Camellia sinensis) against cigarrete smoke-induced mutations (SCE) in humans. J Cell Biochem 1997:27 : 68-75
22. Geer M. Ishaq, Meera S.Cytogenetic evaluation of lemongrass oil (Cymbopogon citratus) using mouse bone-marrow test system.Dissertation submitted to Rajiv Gandhi University of Health Sciences, Karnataka, Bangalore 1999.
23. Chung KT, Wong TY, Huang YW, Lin Y. Tannins and human health; a review. Crit Rev Food Scei Nutr 1998: 38 (6) : 421- 64
24. Sasaki YF,Matsumoto K, Imanishi H,Watanabe M, Tutikawa K.In-vivo anticlastogenic and antimutagenic effects of tannic acid in mice. Mut Res 1990: 244 (1) : 43-7
25. Toering SJ, Gentile GJ,Gentile JM.Mechanism of antimutagenic action of (+) catechin against the plant-activated aromatic amine 4-nitro-o-phenylenediamine. Mut Res 1996: 361 (2-3) : 81-7
26. Gentile JM, Rahimi S,Gentile GJ, Ferguson LR. Effect of selected antimutagens on the genotoxicity of antitumor agents. Mut Res 1998:402 (1-2) : 289-98
27. Elias R, De-Meo M, Balansard G ,Dumenil D. Antimutagenic activity of some saponins isolated from Calendula officinalis L., C. arvensis L. and Hedera helix L. Mutagenesis 1990: 5 (4) : 327-31
28. Lee, B.H., S.J. Lee, J.H. Hui, S. Lee and C.K. Moon (1998) In-vitro antigenotoxic activity of novel ginseng saponin metabolities formed by intestinal bacteria. Planta Med., 64 (6) : 500-3
29. Wilpart M, Speder A, Ninane P, Roberfroid M. Antimutagenic effects of natural and synthetic hormonal steroids. Teratog Carcinog Mutagen 1986: 6 (4) : 265-73
30. Fahrig R.Antimutagenic agents are also co-recombinogenic and can be converted into co-mutagens. Mut Res 1996: 350 (1) : 59-67
31. Guevara AP, Lim-Sylianco C, Dayrit F, Finch P.Antimutagens from Momordica charantia. Mut Res 1990: 230 (2) : 121-6
32. Villasinor IM, Lemon P, Palileo A, Bremner JB. Antigenotoxic spinasterol from Cucurbita maxima flowers. Mut Res 1996: 360 (2) : 89-93
33. Su HY, Cherng SH, Chen CC Lee H.Emodin inhibits the mutagenicity and DNA adducts induced by 1-nitropyrene. Mut Res 1995: 329 (2) : 205-12
34. Zani F, Cuzzoni MT, Dagli M,Mazza P.Inhibition of mutagenicity in S. Typhimurium by *Glycyrrhiza glabra* extract, glycyrrhizinic acid, 18-alpha and 18-beta-glycyrrhetinic acids. Planta Med 1993: 59 (6) : 502-7
35. Schimmer O, Mauthner H. Polymethoxylated xanthones from the herb of Centaurium erythraea with strong antimutagenic properties in Salmonella typhimurium. Planta Med 1996: 62 (6) : 561-4
36. Mitscher LA, Telikepalli H,McGhee H, Shankel DM. Natural antimutagenic agents. Mut Res 1996: 350 (1) : 143-52
37. Choi JS, Lee HJ, Park KY, Ha JO, Kang SS.In-vitro antimutagenic effects of anthraquinone aglycones and naphthopyrone glycosides from Cassia tora. Planta Med 1997: 63 (1) : 11-4
38. Dogasaki C, Murakami H, Nishijima M, Yamamoto K,Miyazaki T. Antimutagenic activities of hexane extracts of the fruit extract and the kernels of Prunus mume. Yakugaku Zasshi 1992: 112 (8) : 577-84
39. Al-Dakkan AA, Al-Tuffail M, Hannan MA.Cassia senna inhibits mutagenic activities of benzo(a)pyrene, aflatoxin B_1, Shamma and methyl-methane-sulfonate. Pharmacol Toxicol 1995: 77 (4) : 288-92
40. Liu TY, Ho KL, Tsai YC, Chiang SH, Chao TW,Chi CW.Modification of mitomycin c-induced clastogenicity by Terminalia catappa L. in-vitro and in-vivo. Cancer Lett 1996: 105 (1) : 113-8
41. Salikhova RA,Poroshenko GG.Antimutagenic properties of Angelica archangelica L. Vestn Ross Akad Med Nauk 1995: 1 : 58-61
42. Emerit, Arutyunyan IR, Lery A, Sarkisian T, Asrian K. Radiation-induced clastogenic factors : anticlastogenic effect of Ginko biloba extract. Free Radic Biol Med 1995: 18 (6) : 985-91
43. Niikawa M, Hayashi H, Sato T, Nagase H, Kito H. Isolation of substances from glossy privet (Ligustrum lucidum Ait.) inhibiting the mutagenicity of benzo(a)pyrene in bacteria. Mut Res 1993: 319 (1) : 1-9
44. Padma PR, Amonkar AJ, Bhide SV. Antimutagenic effect of betel leaf extract against the mutagenicity of two tobacco-specific N-nitrosamines. Mutagenesis 1989: 4 (2) : 154 - 6
45. Sato T, Ose T, Nagase H,Kito H.Mechanism of anti-mutagenicity of aquatic plant extracts against benzo(a)pyrene in the Salmonella assay. Mut Res 1990:241(3) : 283-90
46. Okai Y, Higashi-okai K,Nakamura S.Identification of heterogenous antimutagenic activities in the extract of edible brown seaweeds, Laminaria japonica and Undaria pinnatifida by the Ume gene expression system in Salmonella typhimurium. Mut Res 1993: 303 (2) : 63-70
47. Kohlmeir L,Simonsen N, Mottus K, Neal. Dietary modifiers of Carcinogenesis.Environ Health Perspect 1995:103(8):177-84
48. Block G, Patterson B, Subar A. Fruits, vegetables and cancer prevention: a review of epidemiological evidence. Nutr Cancer 1992: 18: 1-29
49. Krishnaswamy K,Polasa K. Diet, nutrition and cancer – the Indian scenario. Ind J Med Res 1995: 102 : 200-9
50. Poolzobel BL,Bub A,Muller H,Wollowski I, Rechkemnuer G. Consumption of vegetables reduces genetic damage in humans; first results of a human intervention trial with carotenoid-rich foods. Carcinogenesis 1997: 18 (9) : 1847-50
51. Choudhry AR,Das T, Sharma A,Talukder G. Inhibition of clastogenic effects of arsenic through continued oral administration of garlic extract in mice in-vivo. Mut Res 1997: 392 (3) : 237-42
52. Vanboekel MA, Goeptar AR,Alink GM. Antimutagenic activity

of casein against MNNG in the E.coli DNA repair host-mediated assay. Cancer Lett 1997:114 (1-2):85-7

53. Bakalinsky AT,Nadathur SR, Carney AR,Gould SJ.Antimutagenicity of Yogurt. Mut Res 1996: 350 (1) : 199-200
54. Grover IS,Bala S.Studies on antimutagenic effects of guava (Psidium gujava) in Salmonella typhimurium. Mut Res 1993: 300 (1) : 1-3
55. Gruter A,Friederich U,Wurgler FE. Antimutagenic effects of mushrooms. Mut Res 1990:231 (2) : 243-9
56. Samman MA,Bowen ID,Taiba K,Anonins J,Hannan MA.Mint prevents Shamma-induced carcinogenesis in hamster cheek pouch. Carcinogenesis 1998:19(10):1795-801
57. Segaran PP,Delbert MS.Effects of antimutagens on development of drug/antibiotic resistance in micro-organisms. Mut Res 1998: 402 : 139-150
58. McCarty MF.Natural antimutagenic agents may prolong efficacy of human immuno deficiency virus drug therapy. Med Hypotheses 1997: 48 (3) : 215-20.

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Aluminium, Osteodystrophy, Renal

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INTRODUCTION

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multiple Fractures and severe bone pain described in 1960's from Newcastle are Now recognized as " Aluminium Induced Osteodystrophy."

In the present study , the clinical , biochemical, radiological and histopathological spectrum of R.O. D. was studied and analysed along with the role of aluminium in renal bone disease in 50 C.R.F. patients over a period of 1 year.

MATERIAL AND METHODS;-

The material of this study comprised of 50 C.R.F. patients with Male: Female ratio as 1.5:1 and mean age of 48.7 ±18.6 years. The etiology of C.R.F. was chronic glomerulonephritis (CG.N) In 14 patients; diabetic nephropathy in 13 ; benign arteriolar Nephrosclerosis in 10; nephrolithiasis in 5; obstructive and reflux uropathy In 4; polycystic kidneys (P.K.D.) in 2; malignant arteriolar nephrosclerosis In 1 and rapidly progressing glomerulonephritis (R.P.G.N.) in 1. The cases of bone disease due to non-renal causes were not included in this study.

The 50 C.R.F. patients included 3 sub-groups of patients VIZ Predialysis group 37 patients, hemodialysis group 13 (who had been on hemodialysis For a period of 6 months to 2 years (mean o.92 +- 0.64 years) and Aluminium containing phosphate binders (A.C.P.B.) Ingestion group 34 (who had ingested A.C.P.B. from 6 months to more than 5 years.). Each patient was subjected to detailed history, physical examination and investigations including **a complete haemogram; Biochemistry** (urea, creatinine, sodium, potassium, calcium, phosphorus, Alkaline phosphatase, proteins, lipid profile and sugar); **urinalysis; electrocardiogram; Skeletal Survey** (radiographs--skull, wrist, hands, dorsolumbar spine, pelvis, chest and intravenous pyelogram); **Ultrasonography** (kidneys and parathyroid glands); percutaneous renal and iliac crest bone biopsies after informed consent. Bone biopsies were taken from iliac crest using Jamshedi needle (internal diameter 2 mm) under local an anesthesia and cylinders of tissue with length varying from 0.3 cm. to 1 cm. were obtained and fixed in 10% formalin for 24 hours. After dehydration and embedding, the undecalcified sections were stained by H&E stain (Wallington, 1972), a modification of Von- Kossa method (1901) and Aluminon stain[11]. The histopathological diagnosis of bone disease was based on standard diagnostic criteria.

From the patient's data, the following derived quantities were Calculated for each patient :-

1. Creatinine clearance by Cock-Croft formula. (Cock- Croft & Gault, 1977).
   [140- age (years)] x Wt. (kg) x 0.85 (in the female only) 72 x Serum Creatinine Concentration (mg/ dl)
2. Serum calcium concentration corrected for serum albumin below 4 gm/dl. (Agus, et al, 1981). = Calcium (mg/dl) + 0.8.
3. Calcium - Phosphorus Product = Calcium (mg/ dl x Phosphorus (mg/dl).
4. Uremic Index: The total number of signs and symptoms of uremia.[12].
5. Blood urea (mg/dl) to serum creatinine (mg/dl) ratio.
6. Creatinine (mg/dl) - Height Index (cm) (Blackman G.L., et Al, 1977) Actual 24 hour urinary creatinine excretion (mg/dl) = Normative value (for height and sex) of creatine Excreation (mg/dl)
7. Q -Tc Interval (second) in E.C.G. by Bazett's formula:- Q- Tc = Q - T Ö R – R. The data was analyzed using the standard statistical techniques and results were expressed as arithmetic mean ± 1 S.D. The Bayesian methods and student t-test were employed where necessary.

RESULTS

In 50 C.R.F. Patients, the spectrum of clinical positivity was 14% and that of radiological and histopathological positivity 26% and 34% respectively. Aluminium bone disease was seen in 10% of C.R.F. patients and in 29.4% of histopathologically positive R.O.D. patients. Histopathologically, the 17 R.O.D. patients were found to consist of 4-disease patterns viz., osteomalacja (10 patients), osteosclerosis (4), mixed osteodystrophy (3) Aluminium bone disease superimposed on osteomalacia (4) and mixed osteodystrophy (1). Osteomalacia was seen having wide osteoid seams and decreased mineralization; osteoscelrosis as increased total mass and density of bone with more bone per unit area; mixed osteodystrophy as wide osteoid seams, impaired mineralization and peritrabecular fibrosis and Aluminium bone disease as spots of aluminium with no apparent osteoid seams or as dark- back Aluminium lines at osteoid- mineralized interfaces. Osteomalacia constituted the predominant histopathological type of bone disease (58.82%) with predominance of superimposed aluminimum bone disease (23.52%). The mean uremic index in the histopathpologically positive R.O.D. patients was 12.47±2.80. The most frequent bone symptoms and sign were diffuse bone pain (82.35 %), bone tenderness and muscular Weakness (47.05 %) each. Clinically, the predialysis patients had arthropathy (100%), Dwarfish (100%), bone deformities (90%), joint pains (81.8%), cataracts (81.25%), proximal myopathy (80.0%), muscular weakness (80%), bone pain (68.75%) and bone tenderness (62.50%) as the most frequent symptoms and signs. The hemodialysis patients on the other hand, presented with comparatively lower percentage of physical symptoms and signs i.e. waddling gait (50%), bone tenderness (37.50%), and diffuse bone pain (31.25%). The diffuse bone pain had highest sensitivity (82.35%) and specificity (93.93%), dwarfism; Waddling gait and bone tenderness had highest specificity (100%) but low sensitivity (5.88%, 11.76% and 47.05% respectively), for the histological presence of bone disease. Similarly, the positive predictive values of bone pain and waddling gait were 87.50% and 100% respectively and efficiencies of diffuse bone pain and bone tenderness were 90% and 82% respectively. Microcytic hypochromic anemia was seen in 5.88% cases of histopathologically positive R.O.D. Patients, and serum alkaline Phosphatase as values were elevated in 27.27% cases of osteomalacia, 18.18% Osteosclerosis, 18.18% mixed osteodystrophy and 9.09% each in Aluminimum bone disease superimposed on osteomalacia and mixed Osteodystrophy. The highest prevalence of osteomalacia (23.07%) was seen in hemodialysis patients and that of osteosclerosis (8.82%) and mixed Osteodystrophy (8.82%) was seen in patients who had consumed A.C.P.B.

Aluminimum bone disease, as a whole was more prevalent In A.C.P.B. ingestion group (14.70%) but was predominantly more common (10.81%) in predialysis patients having osteomalacia; 21.3% of cases of C.G.N. were associated with Aluminium bone disease. Age-wise, the 11-20 years group had Aluminimum bone disease in 20.00% of patients; 31-40 years in 14.2% and 41-50 years in 33.3%. In diabetic nephropathy, 40% of cases of osteomalacia who had superimposed Aluminimum bone disease suffered from encephalopathy as well; the 20% of which died during the hospital stay and the other 20% were in a moribund state.

The 13 radiologically positive R.O.D. patients consisted of 5 patients (38.46%) having features of osteomalacia (viz. looser's zones, triradiate pelvis, cod-fish vertebrae and fractured vertebrae), 4 patients (30.76%) of secondary hyperparthyroidism (viz. subperiosteal resorption, bone cysts and metastatic calcification) and 4 patients (30.76%) exclusively of osteoporosis; thereby showing osteomalacia as a predominant radiological lesion in this study.

The patients having radiological evidence of osteoporosis had histological evidence of osteomalacia in 30%, osteosclerosis in 10% and Aluminimum bone disease in 20%. Those having features of looser's zone and genu valgum had histological presence of osteomalacia and those having triradiate pelvis, cod-fish vertebrae and osteoslerosis had mixed osteodystrophy.

DISCUSSION

Renal osteodystrophy is a metabolic disease of C.R.F. The long standing alterations in the mineral metabolism generated by renal failure have a profound effect on skeleton and induces a systemic disease[13].

Age-wise, the osseous changes of C.R.F. have been reported in patients having age from 6-90 years.[14] The greater availability of dialysis has eliminated age-limitations leading to an increase in average age of such patients. In the present study, the age of C.R.F. patients included into the study ranged from 14-84 years and the mean age of R.O.D. patients was 47 ± 19.63 years.

Although the male population had predominance over the female during the last census in J&K State - 931 females against 1000 males[15], but the male-female ratio of R.O.D. found in present study was 1 : 1.1, showing predominance in the females.

Of the six districts of Jammu Province, Jammu is the most populous one[15] where greater number of R.O.D. patients (89.32%) were detected in the present study.

In the western countries, the Aluminimum bone disease is found In 50% of unselected cases of C.R.F., in 90% cases of osteomalacia, and 50% in mixed osteodystrophy[16]. The present study showed 10% of unselected cases of C.R.F. having Aluminimum bone disease, 8 % of it superimposed on osteomalacia and 2% on mixed osteodystrophy. Varied prevalence of Aluminimum bone disease has been reported by different authors.

The low prevalence of R.O.D. in tropics differs strikingly from reports in temperate areas and it varies according to centre , region, countries, types and duration of renal pathology, regional sun- light exposure, dietary habits and perhaps the anecdotal variations in bone constitution17. The climate of outer plains of Jammu province is sub- tropical and monsoon type and that of the zone of mountains depends upon the altitude[15,18]. The geographical aspects of the population of patients studied, type of diet and E.S.R.F. therapies received are likely to influence the determination of the histological features of the renal bone disease.

The remediable advancement of the biochemical management of patients with E.S.R.D. (end stage renal disease), dialysis system and increased successful renal transplantation have led to a decline in the frequency and severity of osseous which were among the most serious consequences of renal failure[19]. Mankin[19] reported that the syndrome of R.O.D. is much less common than in prior years.

Coburn,[20] reported low frequency of osteomalacia in areas where the water content is not polluted with Aluminium . Pierdes[21] and Revell Peter A.[22] stated that the commonest feature of R.O.D. in U.S.A. is osteitis fibrosa , while in part of U.K. osteomalacia predominates . In India , Rao M., et al[23] reported 32 cases of R.O.D. out of 50 C.R.F. patients studied and the Aluminium bone disease was seen in 4 cases. In the present study , 17 cases of R.O.D. were detected in 50 cases of C.R.F. and the Aluminium disease was seen in 5 cases. The predominant histopathological type of bone disease was osteomalacia (20%), followed by osteosclerosis (8%) and mixed osteodystrophy (6%). The Aluminium bone disease was found predominantly superimposed on osteomalacia (8 %) and mixed osteodystrophy (2 %).

The Aluminium deposits in bone characteristically induce low turnover type of bone disease i.e. osteomalacia[16,24,25]. In the present study , 14.70 % of patients who had consumed A.C.P.B. for variable duration developed Aluminium bone disease; 8.82 % superimposed on osteomalacia and 5.88 % on mixed osteodystrophy . But 7.69 % of the patients in the hemodialysis group, developed the Aluminium bone disease meaning thereby that the Aluminium bone disease is seen more frequently in A.C.P.B. ingestion group patients and appears to have a strong association with patients consuming A.C.P.B. an observation similar to that reported by Rao[23].

The chief mineral source of Jammu province is Bauxite - an Aluminium ore[15]. Aluminium induces a low turnover type of bone disease i.e. osteomalacia. The present study revealed the majority of R.O.D . patients having osteomalacia with predominance of Aluminium bone disease in patients having osteomalacia histologically. Since the majority of cases of C.R.F. having Aluminium bone disease was seen in A.C.P.B. ingestion group (14.70%), followed by predialysis group (10.8%) and hemodialysis group (7.69%),it can be deducted that the A.C.P.B. ingestion, was the chief source of Aluminium deposition in bones in patients of C.R.F. in present study.

A relationship exists between the body Aluminium accumulation and neurocognitive dysfunction or morbidity in patients of C.R.F[26,27,28]. In the present study, 40% of the patients having Aluminium bone disease had encephalopathy of which 20% died and another 20% were in a moribund state.

Pie[29] studied R.O.D. in 259 diabetic patients and reported that osteitis fibrosa and mixed osteodystrophy were uncommon (8 patients), however aplastic bone disease was more common (19 patients) and Aluminium bone disease was more frequently seen in non-diabetics. In the present study, osteitis fibrosa, mixed osteodystrophy and aplastic bone disease were not seen in the diabetic; however, 3 out of 13 diabetics had osteomalacia, 1 had osteosclerosis and only 2 had Aluminium bone disease superimposed on osteomalacia. So the Aluminium bone disease was seen more commonly in the non-diabetics (3 patients out of 5) in the present study.

In mild and moderate renal failure with G.F.R. less than 40 ml/minute, the woven osteoid is present with . impaired mineralization in some patients[2,16]. Smith[30] stated that in E.S.R.F., all patients have abnormal bone histology and approximately 5% have stainable Aluminium at mineralization front. In the present study, all patients having histopathologically positive R..O.D., had creatinine clearance (or G.F.R.) less than 40 ml/minute with the histological spectrum as: 10 patients of osteomalacia, 4 of osteosclerosis, 3 and 5 of mixed osteodystrophy and Aluminium bone disease respectively.

CONCLUSIONS

The histopathological spectrum of 17 R.O.D patients found in 50 C.R.F. patients studied consisted of osteomalacia in 10 patients, osteosclerosis in 4, mixed osteodystrophy in 3, and Aluminium bone disease superimposed on osteomalacia in 4 patients and mixed osteodystrophy in 1.

All the 5 patients of C.R.F. having Aluminium bone disease had creatinine clearance of less 40 ml/minute and the disease was more commonly seen in non-diabetics; associated with C.G.N.; encephalopathy and in those who had consumed Aluminium containing phosphate binders.

REFERENCES

1. Murli, Sundaram. " Renal osteodystrophy." Skeltal Radiol. (1989). 18: 415-426.
2. Malluche H.H., Maris-Claude Fangere. "Vit. D3 and uremic bone disease." Nephrology Vol. 1. Ed . M. Hitano. 1991.
3. Shinchi, Hosokawa, Atushi Oyamaguchi, Osamu, Yoshida "Tracelements and complications in patients undergoing chronic hemodialysis." Nephron. 1990; 55 : 375-379.

4. Sprague S.M. "Aluminium: Its measurement and metabolism." Semin. Dial. 1988; 1 : 103-111.
5. Chappuis P., de Vernejoul M.C., Paolaggi F., et al. "Relationship Between hair , serum and bone Aluminium in hemodialysis Patients. Clin. Chim. Acta., 1989; 179: 271-278.
6. Charles R. Nolan, Joseph R. Califano and Clifford A. Butzin. "Iinfluence of calcium acetate or calcium citrate on intestinal Aluminium Absorption." Kidney international. Vol. 38 (1990); pp. 937-941.
7. Anon, 1984. "Renel osteodystrophy and status of Aluminium and other trace metals in C.A.P.D. patients." A panel review. Perit Dial. Bull. 4 : 129-136
8. Joffe P., Hyldstrup L., Heaf J. G., Podenpent J. Henriksen J.H. "Biphosphonate Kinetics in patients undergoing C.A.P.D. Relation to dynamic bone histomorphometry , Osteocaqlcin and P.T.H." Am. J. Nephrol. 1992 (6); 410- 24.
9. Andersen J.R., 1987. "Aluminium in renel disease as studied by atomic absorption spectrometry." Thesis. Copenhagen. Page 10-66.
10. Kerr D.N.S., Wallis J., Ellis D.H., Simpson W.P., Undali P. R., Ward M.k. "Bone disease in patients undergoing regular hemodialysis." Journal of bone and joint Surgery. 51 B: 578; 1969.
11. Maloney Norman A. Sussan M. Oyy., Allen C. Alfrey, Nancy L. Miller, Coburn, J.W., Donald J. Sherrard. "histological Quantitation of Aluminium in iliac crest bone patients With renal failure." J. Lab. Clin. Medicine. 1982;90(2).
12. Malluche H.H. " Renal Osteodystrophy." N. Engl. J.Med. 1989; 321 : 317-19.
13. Yu. Y.F., "Pathological analysis of R.O.D. in 194 cases." Chung-hua Ping Li Hsuch Tsa Chih. 1993. Jul., 32 (7); 448-50.18. Kaushal D.S. "The New Rasthriya Atlas." 1993.
14. Malangone J.M., Abuelo J.G., Pezzulo J.C., Lund K., McGloin C.A. "Clin. And lab. features of patients with chronic renal disease at the start of dialysis." Clin. Nephrol. Vol. 31 No. 2. 1989 (77-870.).
15. Raina A.N. "Geography of Jammu & Kashmir." 1993 Ed.
16. Malluche H.H., Fangere M.C. " Renal bone disease 1990 – An Unmet challenge for the nephrologists." Kidney International. 1990; 38 : 193-211
17. Youmbissi T.J., Nai O.R., Gonsu J.F., Blackett K. Ngu, Mbakop A., "Is renal osteodystrophy in rare in tropics?." Nephon. 1990. 50: 227-228.
18. Kaushal D.S. "The New Rasthriya Atlas." 1993.
19. Mankin H.J. "rickets osteomalacia and renal osteodystrophy." The Orthopaedic Clinics of North America." Vol. 21. No. 1. Jan., 1990.
20. Coburn J.W., Norris K.C., Nebeker H.G. (1986). "Osteomalaca In bone disease arising from Aluminium." Semin. Nephr. 6: 68.
21. Pierides A.M., Skillen A.W., Ellis H.A. (1979). " S. Alk. PO_4 in azotemic and hemodialysis osteodystrophy; a study of isoenzyme patterns , their correlation with bone histology and their changes in response to treatment with $1(OH)_2$ and 1, 25 $(OH)_2$ D_3." J. Lab. Clin. Med. 1979;93: 899-909.
22. Revell Peter A . "Pathology bone". 1986 Ed.
23. Rao M., Israel J., Krishnaswamy H., Shastry J.C..M., Jacob C.K. "Renal osteodystrophy in patients with end stage renal disease." Indian Nephrol. New Series . 1993;.3.
24. Ward M.K., Feest T.G., Ellis, H.A., Perkison I.S., Kerr D.N.S., Herrington J., Goode G.L. "Osteomalacia, dialysis osteodystrophy. Evidence for a water borne aetiological agent. probably aluminium." Lancet. 1978; 1 : 1841-1845.
25. Cournot-Wilmer G., Zingraff J., Plenchet J.J., Escaig Infevre R., Bourdeau A., Galle G.M.P., Bourdon M., Druke T., Balson S. "Aluminium localization in bone from hemodialysed patients. Relationship to matrix mineralization." Kidney International. 1981; 20 : 3
26. Chazan, J.A., Libbey N.P., London M.R., Pono L., Abuelo J.G. "The clinical spectrum of R.O.D. in 57 chronic H.D. patients a correlation between biochemical parameters and bone pathology findings." Clin . Nephrol. Vol. 35 No. 2. 1991 (78-85).
27. Sprange S.M., Corwin H.L., Tanner C.M., et al. "Relationship of Aluminium to neurocognitive dysfunction in chronic dialysis patients." Arch . Int. Med. 1988; 148:2169-2172.
28. Maetyn C.N., Barker D.J.P., Osmond D., et al. "Geographical relationship between Alzheimer's disease and Aluminium in drinking water." Lancet 1989; 49-62.
29. Pie. Y., Hercz G., Greenwood C., Segre G., Manuel A., Saiphoo C., Fenton S., Sherrard D., "Renal osteodystrophy in diabetic pateients." Kidney International 1993. Jul., 44 (1); 159-64.
30. Smith A.J., Fangere M.C., Abreo K., Fanti P., Julian B., Malluche H.H. :Aluminium related bone disease in mild and advanced renal failure. Evidence for high prevalence and morbidity and studies on etiology and diagnosis in 197 patients." American J.Nephrol . 1986;6: 275-283.

TREATMENT OF FRACTURES OF THE DISTAL RADIUS WITH EXTERNAL FIXATOR.

Suhail Afzal M.S.; M.R. Mir M.S.; M.A. Halwai M.S.; Shabir Ahmad MBBS

ABSTRACT: *Thirty patients of unstable fractures of distal radius, were treated by closed reduction and external fixator from April 2001 to March 2002 at Govt.hospital for Bone and joint surgery ,consisting of a converging pin configuration and reduced external fixator time.The study consisted of 14 males and 16 females with an average age of 42 years(avg.20-62Years).The patients were followed up for an average of 36 weeks(16-52 weeks). The affected side was right in twenty patients,left side in nine patients and one patient had bilateral involvement.Sixteen patients had an excellent result, nine a good result,three a fair result and two,a poor result,assessed as per the Modified clinical scoring system of Greene and O'Brien(1991)[1]. The two poor results were attributed to the development of Sudeck's Osteodystrophy. Radial length was well maintained,with shortening averaging less than 2mm.*

Key Words:- ***fractures of distal radius, ligamentotaxis, external fixator, percutaneous pinning.***

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Unstable fractures of the distal radius remain a challenging problem despite continued refinements in the treatment. If these fractures are allowed to collapse,radial shortening,angulation and articular incongruity may cause permanent disability. Although some authors have reported satisfactory results with closed reduction and immobilization in cast [2], most have recommended some type of skeletal fixation to maintain the reduction[3,4]. A variety of methods have been devised to avoid loss of reduction, including pinning the distal fragment percutaneously[5], using an external fixator [6,7]. The basic principle in many of these methods is to provide fixed traction which prevents shortening of the radius. The converging pins of the external fixator prevent loosening,thereby diminishing the risks of infection,loss of reduction of fracture, and breakage of pins. This geometry of pins provides rigid fixation even in osteoporotic bone[8].

MATERIAL AND METHOD:

Between April 2001 to March 2002,thirty patients of unstable fractures of distal radius treated in this hospital by external fixator and augmented Kirschner wires percutaneously in some cases were included in this study. The criteria of Cooney et al;[7] were used to determine the unstable fractures of the distal radius.There were sixteen(53%) female and fourteen(47%) male patients, with age range from 20 to 62 years(avg 42 yrs). The right side was involved in Twenty(67%) patients. The left side in nine(30%) patients and one(3%) of the patients had a bilateral involvement. The mechanism of injury was fall on an outstretched hand in twenty eight(93%) patients and motor vehicle accident in two (7%) patients. The average duration from the injury to the operation was two days.

The fractures were classified as per the universal classification(Cooney)[10]. Twelve were extraarticular fractures(type-2),eight were type-3 and ten were type-4 intraarticular fractures. Patients who had an open fracture or an associated fracture of the ipsilateral extremity, were excluded from this study. The patients were assessed clinically and radiologically, before subjecting them for the procedure.

Operative Technique:-

The patients were operated under general or regional anaesthesia. After preparing and draping the limb, the forearm was placed on a side trolley. Closed manipulative reduction was done and confirmed by imaging.A limited open dorsal surgical approach was used for the insertion of 3mm Shanz pins. Two pins were inserted in the radius proximal to the fracture, and two pins were inserted in the second metacarpal at the neck and base.The pins were inserted with there tips pointing towards each other at an angle of 45-60 degrees.The pins were held to the rod with clamps. Final reduction was done and confirmed by imaging and the clamps were tightened, maintaining the traction all along. Kirschner wires were used in cases where some of the fragments were irreducible and in cases were distal radioulnar joint instability was present. At the completion of the procedure, the pin sites were cleaned

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and dressed aseptically.

Post operatively the hand was kept elevated ,the neurovascular status was closely monitored. The patients were instructed excercises for the fingers,elbow and shoulder as per the Codmans six pack system[11].The average stay in the hospital postoperatively was 2 days. At the time of discharge the patients were instructed in pin care and continuation of the physiotherapy programme. Oral analgesics and antibiotics were advised for a period of five days. The patients were followed initially at 2 weeks, and then at 4 weeks. The external fixator was removed at 4 weeks and a below elbow pop cast was applied. The cast and the 'k' wires were removed at 6 weeks, and were advised to continue the physiotherapy of fingers, elbow, shoulder and the wrist. Later the patients were followed up at 6 weeks intervals, till the final assessment was done..

RESULTS:

Twenty eight(93%) patients tolerated the external fixator well. None of our patients got infected.Two(7%) of the patients developed Sudeck's osteodystrophy.Except the two, all patients were pleased with the cosmetic outcome, and were able to return to their regular work and daily activities. Functional assessment was done in terms of pain, functional status,range of motion and grip strength and the points allotted in each one, with maximum of 25 points (Modified clinical scoring system of Greene and O' Brien-1991)[1]. The final results were graded into, excellent(90-100 pts), good(80-89 pts), fair (65-79 pts) and poor (<65 pts).

Pain:

Fourteen(47%) patients had no pain, eleven(37%) had mild occasional pain, three(10%) had moderate but tolerable pain and two(6%) patients had severe and intolerable pain.

Functional status:

Seventeen(57%) of our patients were able to return to their regular work, eleven(37%) patients had restriction of their regular work, and two(6%) patients were unable to work because of pain.

Range of Motion:

Sixteen(53%) of the patients had ROM of 100% of normal, with a dorsiflexion-palmarflexion arc of 120 degrees or more,eleven(37%) patients had ROM of 75-99% of normal , with a DF-PF arc of 91-119 deg,three(10%) patients had ROM of 50-74 % of normal with a DF_PF arc of 61-90 deg.

Grip strength:

Twenty five(83%) patients had a grip strength of 100% of normal,three(10%) patients had 75-99% of normal and two(7%) had grip strength of 50-74% of normal.

On assessing the patients functional outcome based upon the above criteria, the final results were computed as:

RESULTS	NO.OF CASES	%AGE.
EXCELLENT	16	53%
GOOD	9	30%
FAIR	3	10%
POOR	2	7%

DISCUSSION:-

The external fixator provides a simple and reliable means of treating these unstable fractures of distal radius, according to the concept of ligamentotaxis that was proposed by Vidal et al; [12]. If an adequate closed reduction can be obtained, the external fixator maintains the reduction with constant distraction until union occurs. Addition of 'K' wires as a "Joystick" to elevate depressed fragments is effective in restoring a congruent distal radial articular surface [9]. These wires can also be used to obtain stability of distal radio-ulnar joint, which is most of the times affected in distal radial fractures.

The functional result is the most important clinical parameter when assessing the results of the treatment of distal radial fractures [7]

Many authors recommend shorter fixation periods [13]. Our concern was that reducing the fixation period might increase the risk of redisplacement of the fracture and this might spoil the functional results, but our data does not suggest so.We also computed the radial shortening as the difference between the intial post-reduction and final roentgenograms, made for each patient.There was an average loss of 2mm, which is in consonance with the other published series[7].

We feel that the high rate of excellent and good results, and the low rate of complications in our study were influenced by the fact that all the fractures were closed, without additional injuries of the ipsilateral extremity, inherent stability of the converging pins [14,15], reduced time of external fixator and strong patient motivation to regain early and complete function.

In our study, the data suggests that external fixator with augmentation by 'K' wires as and when indicated, is a satisfactory method of treating the fractures of the distal radius and the outcome is likely improved with shorter duration of external fixator application.

References:-

1. Cooney,WP; Saffar H;: Fractures of distal radius-Martin Dunitz 1995,pp 124.
2. Spira,E;Weigl,K:- The communitted fractures of distal end of radius.Reconstruction Surgery and Traumatology 1969,:11:128-

138.

3. Palmar AK: Fractures of distal radius.Operative hand Surgery.Ed2.Vol:2.pp 1006-1007.New york,Churchill livingstone.1988.
4. Melone CP Jr: Open treatment for displaced articular fractures od distal radius.Clin.orthop 1986: 103-111;202.
5. Depalma AF;- Communitted fractures of distal radius treated by ulnar pinning.JBJS[Am] 1961:43A ,657-668.
6. Anderson R; O'Neil G: communitted fractures of distal radius; Surg,Gyn/Obs:1944:78:434-440.
7. Cooney WPI11; Lindscheld RL; Dobyns JH:- External pin fixation for unstable colle's fractures.JBJS[Am] 1979;61A pp 840-845.
8. George S; Edwards JR;- Intraarticular fractures of distal radius treated with small AO fixator;JBJS[73A] Sept.1991.
9. William H Seitz,Jr et al:- Augmented external fixation of unstable distal radius fractures;Journ.of Hand Surgery. 1991;16A(6).
10. Cooney, WP:- Symposium on management of intraarticular fractures of distal radius:Cont.Othop,Vol 21.July 1990.
11. Cooney,WP: Saffar PH:- Fractures of distal radius.1995.
12. Vidal J;et al:- Une methode orginale dans le traitment des fractures.Acta Orthop Belgica,1977: 43:781-789.
13. Majed Haddad,et al:- External fixation of distal radius fractures, 3 or 5 weeks of external fixation.Inst.Orthop[SICOT] 2000,21:224-226.Springer-Verlag.
14. Evans N: Harris JD; Kenwright J; Grew ND:Tanner KE:- The oxford external fixateur .Sixth annual report of the Nuffield orthop centre,Headington,Oxford.1979.
15. Chao EYS; Pope MH:- The mechanical basis of external fixation. In concepts in External fixation,pp 13-39.Edited by Seligson and Malcolm pope.New York.Grune and Stratton.1982.

ENTEROCOCCAL INFECTIONS : AN OVERVIEW OF SKIMS SCENARIO

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Abstract: Two hundred and fifty of pathgogenic streptococci were isolated form PUS (75), Throat (50), Urine (50), blood (10) and CSF (4). The highest isolation was of group A streptococci (71 strains) followed by enterococci (70 strains). Enterococci are associated with both community and hospital acquired infections. Patients on multiple antibiotic make enterococci able to survive and proliferate. The cause being underlying disease, prolonged hospital stay, prior surgery, renal insufficiency and physical intervention like urinary and vascular catheters and residency in intensive care unit settings.

Key words: Infection, enteraocci, streptococci.

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Introduction:

The past few years have witnessed increasing interest in enterococci. Until recently, these ordinary bowel commensals languished as misclassified streptococci, commonly perceived with the exception of endocarditis and rare cases of meningitis ... (as) not ... a major cause of serious infection[1] in the last decade, however, enterococci have become recognized as leading causes of nosocomial bacteremia surgical wound infection and urinary tract infection[2,3]. Two types of enterococi cause infection 1. Those originating from patients native flora, which are unlikely to possess resistance beyond that intrinsic to the genus and are unlikely to be spread from bed to bed. 2). Isolates that possess multiple antibiotic resistance traits and are capable of nosocomial transmission.

Enterococci are exceedingly hard . They tolerate a wide variety of growth conditions, including temperatures of 10°C to 45°C and hypotonic, acidic or alkaline environments. Enterocci are intrinsically resistant to many antibiotics. Unlikely acquired resistance and virulence traits, which are usually transportation or plasmid encoded, intrinsic resistance is based in chromosomal genes which typically are nontransferable.

Material and Methods:

This study was conducted in the department of Microbiology, SK Institute of Medical Sciences, Srinagar on patients (both outdoor and indoor) who attended the hospital for a period of two years. Patients suspected of having sore throat, tonsillitis, pharyngitis, puerperal sepsis, septicemia, wound infections, respiratory infections and other systemic infections were taken. Patients of all age groups were included in the study. All specimens were processed within 1 hour of their receipt in laboratory. In addition to routine blood and chocolate agar, selective streptococcal agar and crystal violet blood agar plates[4] were used for the selective culture of streptococcus as these medias inhibit the growth of staphylococci and some other bacteria found in clinical specimens.

The plates were incubated in a candle jar with 5-10% CO_2 at 37°C for 18-24 hours. Identification of colonies was done followed by antibiotic sensitivity.

Serotyping of colonies was done by latex agglutination kit method (Mathur NB 1991) kit manufactured by Rashmi enterprises).

Observation:

Two hundred and fifty isolated of pathogenic streptococci were recovered from the following sources PUS (75), Throat(50), Urine (50), blood (10) and CSF (4).

Sources of isolation of enterococci according to age

Total No. of enterococci	Specimen	0-4 Yrs	5-18 Yrs	16-80 Yrs	31-40 Yrs	Above 41 yrs
70(28%)	PUS (36)	3(1)	5(5)	7(5)	11(10)	10(4)
	Urine (28)	2	-	10(5)	7(2)	9(3)
	Vaginal swab(2)	-	-	-	2(2)	-
	Sputum (2)	-	-	-	-	2(2)
	Blood (2)	-	-	-	-	2 (Immunosuppressed)
	Total	5	5	17	20	23
	70	(7.1%)	(7.1%)	(24.2%)	(28.5%)	(32.8%)

The number in parenthesis shows number of enterococci in mixed culture with klebsiella, staphylococcus aureus and pseudomonas. Out of 70 isolates of enterococcus maximum isolation from pus 36(51.4%) and urine 28(40%). Maximum age involved 41 yrs and above 32.8%.

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Isolation of streptococci with their respective sources and antigenic groups

Clinical specimen	No of strains of streptococci	A	B	C	G	F	Enterococci	Strep Virdians	Strep Pneumonia
Pus	75	25 (33.3%)	2 (2.6%)	5 (6.6%)	3 (4%)	3 (4%)	36 (48%)	-	-
Urine	50	9 (18%)	7 (14%)	4 (8%)	2 (4%)	-	28 (56%)	-	-
Throat swab	50	27 (54%)	7 (14%)	7 (14%)	5 (10%)	2 (4%)	- (8%)	-	2 (4%)
Vaginal swab	25	6 (24%)	13 (52%)	1 (4%)	2 (8%)	1 (4%)	2 (8%)	-	-
Sputum	36	2 (5.5%)	1 (2.7%)	1 (2.7%)	-	-	2 (5.2%)	-	30 (3.3%)
Blood	10	1 (10%)	1 (10%)	-	-	-	2 (20%)	4 (40%)	2 (20%)
CSF	4	-	1 (25%)	-	-	-	-	-	3 (75%)
Total	250 (100%)	71 (28.4%)	32 (12.8%)	18 (7.2%)	12 (4.8%)	6 (2.4%)	70 (28%)	4 (1.6%)	37 (14.8%)

The majority of isolates belonged to group A 71(28.9%) followed by enterococci 70(28%). Association between isolation of isolates of streptococci from respective sources is significant as p value is <.001.

Enterococci were frequently found as part of mixed gram positive and gram negative flora in these settings.

Source of Isolation of streptococci / enterococci according to Diagnosis

Specimen	Clinical diagnosis	No. of Strains	Enterococci
PUS (75)	Wound abscess	35	12
	Py. dermatitis	14	8
	Diabetic ulcers	5	5
	Discharging sinus	7	6
	Umbilical sepsis	5	5
	Miscellaneous	9	-
Urine (50)	Rec. UTI	29	15
	Pyelonephritis	7	5
	Renal stones	4	3
	Diabetes mellitus	4	2
	Hypertension	3	-
	Toxemia of pregnancy	3	3
Throat swab (50)	Tonsillitis	22	-
	Pharyngitis	11	-
	Arthritis	6	-
	Bronchopneumonia	11	
Sputum (36)	Pneumonia	18	-
	Lung abscess	4	-
	RHD with valvular lesions	5	-
	CA lung	2	2
	Chronic renal failure	4	-
	Miscellaneous	3	-
Vaginal swab (25)	Puerperal sepsis	12	1
	Pelvic inflammation	6	1
	Amnonitis	6	-
	Miscellaneous	1	-
Blood (10)	Septicemia	7	2
	Endocarditis	3	
CSF (4)	Meningitis	4	-
	Tolal	250	70

Discussion:

[illegible] prevalence of group A streptococci (28.4%) was [illegible] our study, but it was immediately followed by [illegible] species (28%). The higher prevalence of enterococci is because the study is hospital based and since enterococci are resistant to multiple antimicrobial agents, this allows them to survive and proliferate in patients receiving antimicrobial chemotherapy. This certainly accounts for their ability to cause super-infections in patients receiving a number of different broad-spectrum antimicrobial agents. This leads to increased morbidity and mortality increased and prolonged hospitalization, use of remaining effective antibiotics and leading to greater selective pressure, which further increases the antibiotic resistance. Enterococci survive heat and desiccation, can survive in the environment for prolonged period of time. Health care workers also provide the potential for spread. Inside hospitals the emergence of enterococci was facilitated by practices that allowed the organism to instantly spread from one susceptible human to another, thermometers, catheters and surgical instruments, I/V lines, mechanical ventilation and over use of cephalosporin types antibiotics[5,6].

An accurate and early diagnosis of all the serographs of streptococci should be done so as to prevent the delayed sequlae and check the transmission of disease in hospitals and also eliminate the indiscriminate use of antibiotics. In conclusion enterococcus is emerging as a nosocomial pathogen and care should be taken in hospitals and a well-outlined strategy should be laid including a) injection prevention b) eradicating infections c) proper antibiotic usaged) and preventing the transmission which will reduce the incidence of enterococci.

References:

1. Murray BE. The life and times of the enterococcus. Clin. Microbiology Rev. 1990;3:46-65.
2. Moellering RC Jr. Emergence of enterococcus as a significant pathogen. Clinc. Infections Disease 1992;14:1173-8.

3. Schaberg DR, Culver DH, Gaynes RP. Major trends in the microbial etiology of nosocomial infection. Am J Medicine 1991;91:121-5.
4. Oliver F Roddey, Herbert W Clergg. Comparison of throat culture methods for the recovery of group A streptococci in a paediatric office setting. JAMA 1995;274:1863-1865.
5. Livornese LL, Jr et al. Plasmids, drug resistance and gene transfer in the genus streptococcus. Annuals of Internal Medicine 1992;117:112-116.
6. Pallares R, et al. Vancomycin resistant enterococci. Archieves of Internal Medicine 1993;153:1581-86.

THE ROLE OF ULTRASONOGRAPHY IN BLUNT ABDOMINAL TRAUMA: A RETROSPECTIVE STUDY

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Abstract : *This retrospective study assessed the ability of abdominal ultrasound (USG) to confirm the presence of abdominal or pelvic injury as an indicator of the need for emergency laparotomy in 56 cases of Blunt Abdominal Trauma (BAT), with surgical findings used as diagnostic standard. It also summarised the recent literature on the use of ultrasound in evaluating blunt abdominal trauma as an adjunct or an alternative to diagnostic peritoneal lavage (DPL) and abdominal computed tomography (CT).*

Keywords : *Ultrasound (USG), Blunt Abdominal Trauma (BAT), Diagnostic peritoneal lavage (DPL) , Computerised Tomography (CT)*

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Introduction :

The diagnosis of intra-abdominal injury following blunt abdominal trauma presents a diagnostic challenge even to the skilled surgeon. Clinical findings are often equivocal even as failure to detect injury may lead to significant morbidity and mortality [1, 2], while unnecessary surgery has to be avoided in patients who can be managed conservatively. Expedient and accurate diagnosis is thus essential in the evaluation and management of patients with blunt abdominal trauma suspected of having intra-abdominal injury [3]. These patients can benefit from the high level of accuracy of abdominal ultrasound as the primary screening modality as it is accurate, noninvasive, rapid and portable, and allows serial examinations to be easily performed.

Materials And Methods :

This retrospective study was conducted in 56 consecutive patients of blunt abdominal trauma admitted in the Surgical Casualty Department of S.M.H.S Hospital, Srinagar, Kashmir who were initially evaluated by ultrasound and later underwent surgical intervention based on the USG findings or because of deteriorating clinical status and suspicion of intra-abdominal injury despite negative USG findings.

Radiologists from the Department of Radiodiagnosis and Imaging performed the examinations using a standardized procedure on a bedside USG unit (RT 3200; GE) with a 3.5 MHz convex probe. Patients were scanned with their bladders filled, either before bladder catheterization, or after temporary clamping of the catheter or filling with 200-300 ml of sterile saline through a Foleys catheter. USG examination included the four abdominal quadrants, the suprapubic region, and the subxiphoid region and assessed for visceral injuries and free fluid in the abdomen or pelvis with particular reference to fluid in the Morrison's pouch [4]. USG findings were considered positive if free fluid was present or parenchymal abnormalities consistent with trauma were detected and and in each case, US findings were correlated with those of subsequent surgical intervention. Data recorded included demographics, mechanism of injury and USG and operative findings.

Results:

The ages of 56 patients in our study population ranged from 3 years to 54 years, with the mean age being 34.2 years. Eleven patients belonged to the pediatric age group of 12 years or less. 43 patients were male and 23 patients were female, with the male: female ratio being 1.9:1 . USG findings were positive in 51 patients. **False-negative** findings included four cases of subtle visceral injuries (1 splenic and 3 hepatic contusions) not detected by USG, and one patient who had significant amount of free fluid on laparotomy which was not picked up by USG due probably to sub-optimal operator technique. In two cases the initial USG failed to depict small amounts of fluid and in both the cases free fluid was identified with repeat USG examination with full bladder and confirmed by the surgical findings and these cases have been included as positive cases.There were no patients with **false-positive** findings of free fluid or parenchymal injury. **Sensitivity** of US was 91 %(51 of 56 patients) and **Specificity** of US was 100 % (51 of 51 patients). **Positive predictive value** was 100 % and **overall accuracy** was found to be 93 %.

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Table 1. Causes of BAT

Total No. of patients	56
Road Traffic Accident (RTA)	29 (51.8%)
Blow to abdomen	13 (23.2%)
Fall from height 11	(19.6%)
Injury by an animal 03	(5.4%)

Table 2. Operative Findings

Total No. of patients	56
Splenic injury	29 (51.8%)
Liver injury	17 (30.3%)
Renal injury	05 (8.9%)
Bladder injury	02 (3,6%)
Others	03 (5.4%)

Discussion :

While the use of **USG** in the management of abdominal trauma has been extensively studied in Europe and Asia since the early seventies, it has only recently been accepted as an emerging modality in the United States where **DPL**, as a time-honored test, and **CT**, as the current diagnostic standard, are the primary modalities used[1,6,7].

USG has the advantage of being accurate, noninvasive, rapid, portable and repeatable. DPL is fast and accurate but is associated with complications and cannot be performed in patients with recent laparotomy, pregnancy, coagulopathy etc. CT is also accurate but time-consuming, and requires stability and transportability of the patient, besides being quite expensive in our set-up.

A review of the literature pertaining to the use of ultrasound in the evaluation of patients with blunt abdominal trauma confirms the high level of accuracy of USG and recommends its use as the primary screening modality in BAT. The **sensitivity, specificity, positive predictive value, negative predictive value**, and **overall accuracy** of US for identifying abdominal or pelvic free fluid were found to be **69%, 100%, 100%, 95%**, and **95%**, respectively in a comprehensive study conducted by **Nunes et al** in 2001[3]. **Dolich** and colleagues (2001) [8] observed that US had a sensitivity of 86%, a specificity of 98%, and an accuracy of 97% for detection of intra-abdominal injuries. Positive predictive value was 87% and negative predictive value was 98%. According to a study conducted by **Brown** and co-workers (2001) [9], specificity of US was 96% (2,429 of 2,521 patients), and overall accuracy was 96% (2,574 of 2,693 patients). Positive predictive value was 61% (145 of 237 patients), and negative predictive value was 99% (2,429 of 2,456 patients). Similar findings had earlier been reported by various researchers [2,3,8,10-14]. Many researchers have also recommended replacement of DPL and CT by USG in the initial management of BAT[8,12,15]. The cost-effectiveness of USG viz-a-vis DPL and CT has been reported [15]. The findings of our study correspond well with the established literature.

Conclusion :

Abdominal Ultrasound (USG) performed by experienced operators allows fast and accurate screening for injury in patients with blunt abdominal trauma. The presence of free fluid or parenchymal injury is a highly reliable indicator of the need for emergency laporatomy. In the absence of free fluid or parenchymal injury on USG, conservative management for stable patients is recommended. Diagnostic peritoneal lavage (DPL) or Computerised Tomography (CT) should be initially performed only if USG is not available.

References :

1. McElveen TS, Collin GR. The role of ultrasonography in blunt abdominal trauma : a prospective study. Am Surg 1997 Feb;63(2):184-8
2. Nordenholz KE, Rubin MA, Gularte GG, Liang HK. Ultrasound in the evaluation and management of blunt abdominal trauma. Ann Emerg Med 1997 Mar;29(3):357-66
3. Nunes LW, Simmons S, Hallowell MJ, Kinback R, Trooskin S, Kozar R. Diagnostic performance of trauma US in identifying abdominal or pelvic free fluid and serious abdominal or pelvic injury. Acad Radiol 2001 Feb;8(2):128-36
4. Jehle D, Guarino J, Karamanoukian H. Emergency department ultrasound in the evaluation of blunt abdominal trauma. Am J Emerg Med 1993 Jul;11(4):342-6
5. Kristensen JK, Bueman B, Kuhl E. Ultrasonic scanning in the diagnosis of splenic hematomas. Acta Chir Scand 1971; 137:653-657.
6. Amoroso TA. Evaluation of the patient with blunt abdominal trauma: an evidence based approach. Emerg Med Clin North Am 1999 Feb;17(1):63-75
7. Givre S, Kessler S. The evaluation of blunt abdominal trauma: the evolving role of ultrasound. Mt Sinai J Med 1997 Sep-Oct;64(4-5):311-5
8. Dolich MO, McKenney MG, Varela JE, Compton RP, McKenney KL, Cohn SM. 2,576 ultrasounds for blunt abdominal trauma. J Trauma 2001 Jan;50(1):108-12
9. Brown M, Casola G, Sirlin C, Patel N, Hoyt D. Blunt abdominal trauma: screening us in 2,693 patients. Radiology 2001 Feb;218(2):352-8
10. Bode PJ, Niezen RA, van Vugt AB, Schipper J. Abdominal ultrasound as a reliable indicator for conclusive laparotomy in blunt abdominal trauma. J Trauma. 1993 Sep;35(3):492
11. Lingawi SS, Buckley AR. Focused abdominal US in patients with trauma. Radiology 2000 Nov;217(2):426-9
12. McKenney M, Lentz K, Nunez D, Sosa JL, Sleeman D, Axelrad A, Martin L, Kirton O, Oldham C. Can ultrasound replace diagnostic peritoneal lavage in the assessment of blunt trauma? J Trauma. 1995 Aug;39(2):393-5
13. McKenney MG, Martin L, Lentz K, Lopez C, Sleeman D, Aristide G, Kirton O, Nunez D, Najjar R, Namias N, Sosa J. 1,000 consecutive ultrasounds for blunt abdominal trauma. J Trauma 1996 Apr;40(4):607-10; discussion 611-2
14. Singh G, Arya N, Safaya R, Bose SM, Das KM, Khanna SK. Role of ultrasonography in blunt abdominal trauma. Injury 1997 Nov-Dec;28(9-10):667-70
15. Arrillaga A, Graham R, York JW, Miller RS. Increased efficiency and cost-effectiveness in the evaluation of the blunt abdominal trauma patient with the use of ultrasound. Am Surg 1999 Jan;65(1):31-5

INGESTED FOREIGN BODIES - A PROSPECTIVE STUDY

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Abstract: *A prospective study of 220 indoor patients with ingested foreign bodies was carried in the department of Otorhinolaryngology and Head and Neck Surgery Govt. Medical College Srinagar from May 2000 to May 2002.Males were more commonly involved than the females in the ratio of 1.3:1.The most commonly involved age group was less than 10 years (40.45%). The most commonly removed foreign body was coin in less than 10 years of age (82%) and meat bone in those who were above 10 years of age (53.43%). The commonest site of foreign body impaction was cricopharynx (77.72%). Most of the foreign bodies were radio-opaque (81.8%). There was neither any case of complication following removal of foreign bodies nor there was any case of anatomical abnormality or lesion in-patients with oesophageal foreign bodies.*

Key words: *Foreign bodies, coins, meat bones, cricopharynx.*

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Introduction:

Ingested foreign bodies in the upper aero-digestive tract form significant part of the "On Call" activities of the ENT staff(upto 30% of out-of hour referals)[1]. Dealing with these foreign bodies can be difficult and failure to recognize or remove them promptly can lead to morbidity and mortality[2]. Foreign bodies are most common in children, the elderly, those with dentures or with local disease of the pharynx or oesophagus [2,4] .Hypopharynx, upper oesophagus and oropharynx have been confirmed as sites of impaction [1,5]. Foreign bodies in the hypopharynx and upper oesophagus are common in our experience (77.72%). Radiographs are frequently performed and are usually helpful (81.8%). The localization of foreign body in the upper aero-digestive tract by a patient is also of great importance and needs endoscopic examination even if signs of foreign body are not seen on a radiograph[6].

Material and methods:

A prospective study was undertaken on 220 patients admitted in the department of ENT and Head and Neck Surgery with a recent history of foreign body ingestion from May 2000 to April 2002. A proforma was used to record patient's information at presentation, and the outcome of investigations and treatment. In particular, patient's age, sex, symptom at the time of presentation, type and site of foreign body found was noted. All patients were advised to obtain a roentgenogram of soft tissue neck (lateral and Antero-posterior view) and roentgenogram of chest (Antero-posterior) when required. In case of coin ingestion, if foreign body was not visualized in X-ray chest, roentgenogram of abdomen (AP view) in standing posture was advised. Where possible the foreign body was removed using topical local anaesthetic agent. Patient in whom this was not possible, and where a foreign body was considered to be present on clinical or radiological grounds, endoscopy was done under general anaesthesia and the site of foreign body at the time of removal again noted. Those patients where foreign body was not suspected on clinical or radiological grounds in the upper digestive tact were not included in the study. Barium swallow was advised in those patients where an anatomical abnormality, lesion or a pouch was suspected.

Results:

A total of 220 patients, wherein an ingested foreign body was removed, were studied over a period of two years from May 2000 to April 2002.

Age & sex distribution: The age at presentation ranged from one (1) year to 70 years. The most common age group with a foreign body was <10 years. However, the distribution of ages where a foreign was removed was spread across all age groups (Table I). Males were more commonly involved than females in the ratio of 1.3:1.

Nature of ingested foreign body: The types of foreign body found were 93 coins (42.27%), 70 meat bones{ (31.81%) including sheep, beef and poultry bones}, 25 meat boluses(11.36%), 12 dentures(5.45%), 9 fish bones(4.09%) and 11 others including pins, needles, hooks etc. (Table II)

Site of impaction: All foreign bodies removed were proximal to oesophageo-gastric junction. Out of total 220 foreign bodies removed, 171 (77.73%) were found in cricopharynx and upper oesophagus. Other sites were mid-oesophagus 29 (13.19%), pyriform fossae 9 (4.10%).

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posterior pharyngeal wall and tonsils 4 each (1.81%each) valleculae 2 (0.90%) and lower oesophagus one (0.45%) - Table.IV

Management: After clinical assessment one of the following courses of action was decided upon. 9 foreign bodies were removed at the time of initial clinical examination from the tonsils, posterior pharyngeal wall (4-each) and vallecula (1) using a tongue depressor, illumination and a pair of forceps. These included eight fish bones and one steel wire. In the rest 211 endoscopies were performed under general anaesthesia and foreign bodies removed. Only those endoscopies were included in this study where a foreign body was removed.

Radiographic examination: The radiographs of the soft tissue neck (lateral view) and P-A view chest radiograph (where applicable) were advised after clinical assessment. In the case of coin, meat bones, metallic foreign bodies and fish bones, radiographs were diagnostic (81.8%). In the presence of symptoms where a foreign body was not visualized on radiograph, endoscopy was still done. Radiographs for other foreign bodies were helpful in confirming or revealing the presence of a foreign body. Three radiographic signs were found to be helpful in such cases; presence of a foreign body shadow, gas in the upper oesophagus and increased depth of the prevertebral soft tissue shadow.

Discussion:

In this study patients were in the age range of 1-70 years with no age group exemption. Younger children below 10 years of age were more prone to foreign body ingestion (40.91%) and coin was the most common foreign body in this age group (82.02%) This is in consistent with the findings of Ellen[2] This may reflect the easy availability of coins and exploring the nature of an object by putting in the mouth in younger children. Other foreign bodies including meat bones, meat boluses and dentures were more common above 10 years.This is consistent with the findings of Jone's et-al[7]. This is because of the differences in eating patterns such as taking more meat in this part of the country. Males were more commonly involved than females in the ratio of 1.3:1.This is consistent with the findings of Flynn[1]

The role of radiograph in coin ingestion, meat bones, metallic foreign bodies and fish bones is definite. But 17% of radiographs for ingested foreign bodies could be avoided if careful history and proper examination of the cricopharynx and hypopharynx is performed[1]. In other cases clinical features such as pain, dysphagia or pooling of saliva will indicate the suspicion of a foreign body 211 endoscopies were performed under general anaesthesia to remove foreign bodies. There was no complication. This is probably because these endoscopies were performed by experts. No anatomical abnormality has been noted in patients with oesophageal foreign bodies in comparison to other studies where upto 22% of patients had underlying anatomical abnormality[2].

Table I: Age distribution of the patients.

Age in years	No. of foreign bodies	%age
< 10	90	40.91
11-20	44	20.00
21-30	33	15.00
31-40	24	10.91
41-50	16	7.28
51-60	9	4.09
61-70	4	1.81
TOTAL	**220**	**100.**

Table II: Foreign bodies-type & %age

Type of foreign body	Number	%age
Coin	93	42.27
Meat bone	70	31.81
Meat bolus	25	11.36
Denture	12	5.45
Fish bone	9	4.09
Steel wire	4	1.81
Safety pin	2	0.90
Drawing pin	2	0.90
Metallic hook, ear ring & Syringe Needle	1 each	0.45 each
Total	**220**	**100**

Table III:Type & percentage of foreign body <10 years

Type of foreign body	Number	%age
Coin	73	82
Meat bone	9	10.14
Meat bolus	2	2.26
Safety pin	1	1.12
Denture	1	1.12
Metallic hook	1	1.12
Ear ring	1	1.12
Syringe needle	1	1.12
Total	**89**	**100**

Table IV: Site of impaction & percentage

Site	No. of foreign bodies	%age
Cricopharynx	171	77.73
Mid oesophagus	29	13.19
Pyriform fossae	9	4.10
Posterior pharyngeal wall	4	1.81
Tonsils	4	1.81
Valecullae	2	0.09
Lower oesophagus	1	0.46
Total	**220**	**100**

References:

1. P.O Flynn & R Simo. Fish bones and other foreign bodies. Clinical Otolaryngol 1993, 18,231-233
2. Ellen M Friedman. Tracheobronchial foreign bodies.

Otolaryngologic Clinics of North America Feb. 2000,33(1), 179-185

3. Martin Caravati, Dion L Bennett, Newell E.Mclwee.Pediatric coin ingestion. AJDC 1989,143.549-551
4. N umapathy, J panesar, B.F Whitehead, J.F.N Taylor.Removal of foreign body from the bronchial tree. The Journal of Laryngology and Otology Sep. 1999,113,851-853
5. P.J Nash. A foreign body in the larynx. J Anaesth. 1976, 48,371-372
6. A.A.P Connolly, M Birchall, G.P Walsh & V Moore-Gillon. Ingested foreign bodies.Clin. Otolaryngol.1992, 17,520-524
7. N.S Jones, F.J Lannigan & N.Y Salama.Foreign bodies in the throat. Jr. of Laryngology & Otology 1991,105, 104-108

CLINICAL ASPECTS OF OBSTRUCTED LABOUR

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Abstract: *Three hundred and seventy cases of obstructed labour were seen from 01-11-1989 to 30-04-1991 out of total confinements of 16,403 during the said period giving an incidence of 2.24% in Kashmir Valley. Contracted pelvis & CPD was seen to be commonest cause of Obstruction (59.18%) followed by Malpresentation (36.39%). There were 27 cases of rupture uterus in the series giving an incidence of 7.29%. Maternal and prenatal mortality has been 2.16% and 37.83% respectively. 4 cases of vesico-vaginal fistulae were recorded in the present series. Intelligent anticipation during antenatal check-up and timely referral to well equipped centers is suggested to prevent occurrence of obstructed labour. The basic management of obstructed labour is urgent delivery to avoid further distension and rupture of uterus and to salvage the foetus if it is still alive.*

Key words: *Obstructed labour, contracted pelvis, CPD, Malpresentation*

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Introduction

Any woman who is unable to deliver by natural means is said to be in obstructed labour[1]. These cases are usually un-booked brought late in labour with rupture membrane, infected and in a state of exhaustion and some report with rupture of uterus usually handled by unskilled midwives. The magnitude of Perinatal and maternal complications, which result from occurence of obstructed labour, has given the necessary stimulus for present study. The study under review has been conducted in Lalded Hospital, a teaching Maternity Hospital associated with Government Medical College, Srinagar. Three hundred and seventy cases of Obstructed labour were seen during a period of one and a half year from 1-11-89 to 30-4-1991. An attempt has been made to find out major causes and the incidence of obstructed labour in Kashmir valley, its relation with age, parity , stature and social economic status of the patient. Different consequences and Perinatal and maternal mortality has been analyzed.

Observation

There were total of 16,493 confinements during this period 370 cases of obstructed labour were seen for the same period giving an incidence of 2.24%. Majorities of patients with obstructed labour were in the age group of 21-30 years (59.45%) followed by 31-40 years (25-40). Out of these 77.3% were of rural origin while 22.7% belong to urban areas. Two hundred and fifty patients belong to a poor economic group with a monthly income of less than Rs. 600/-. Patients from rural areas were un-booked and most of these patients (70.27%) were handled by Dais at home and gave history of receiving drugs in the form of injection and IV infusion. In urban group of 84 patients, 30 were booked (35.71%) 247 patients (66.74%) did not attend hospitals for socio economic factors and because of ignorance another sizeable number of patients (18.37%) could not attend the hospital in view of curfew restrictions which were quite often imposed in the valley during that period of turmoil (27.29%) were primigranda 52.97% were of para 1-4, 19.74% and were para. The height was less than 5ft 2" in 296 cases.

Clinical Presentation

All the patients in the present series were having Tachycardia, 301 cases (81.35%) were having signs of dehydration. Distended bladder was seen in 83.78% cases Bandl's ring observed in 86 cases (23.24%). Membranes were absent in 315 patients (85.13%). 246 cases (69.18%) had associated problems in the form of anaemia 196 cases. Osteomalacia 21 cases PET in 4 cases, Diabetes in 3 patients and APH in 4 cases. Contracted pelvis and CPD were seen in 219 cases (59.18%) to be the cause of obstructed labour followed by mal presentation (35.39%) out of mal presentation transverse lie was seen to be commonest cases of obstruction (22.70%). Foetalmal formation was seen in 10 cases (2.70%).

Method of Delivery

311 patients (84.05%) were subjected to caesarian section while in 27 patients (7.29%) had vacuum extraction. Out of patients who underwent caesarian section wound sepsis was seen to be commonest complication 98 cases (31.59%) followed by post-operative shock, 70 cases (22.50%) PPH was seen in 50 patients (16.07%) and extension of incision in 39 cases (12.54%). Out of 27 patients were caesarian Hysterectomy was done wound sepsis was seen in 29.625%, post-operative shock in 37.03% and Peritonitis 18.51%. 32 patients who were delivered vaginally PPH was seen in 28.12%. wound sepsis

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in 37.5% and complete perineal Tear in 2.37% of cases. Cervical tears was seen in 2 patients. There were 27 cases of rupture uterus (7.29%) tranverse lie with impacted shoulder was seen in 15 patients and scar rupture was seen in 7 cases. There was history of receiving oxytoxic drugs at home in 13 patients. In 229 cases were foetal head was impacted for a long time., indwelling catheter was kept for 10 days as pressure necroses of bladder was likely, despite of this 4 cases of VVF were recorded.

Maternal and Perinatal Mortality

There were total of eight maternal deaths in the present series of 370 cases of obstructed labour (2.16%). 5 patients died of septcemia, 2 patients due to hypovolumic shock while one patient died of disseminated intravascular coagulopathy. 140 cases of perinatal deaths were observed in the present series, 64 were still birth while 56 cases were neonatal deaths.

Discussion

In advanced countries modern obstetrics care and rarity of severally contracted pelvis have led to virtual disappearances of obstructed labour. But in developing countries like in India obstructed labour still remains a frequent obstetric complication. During the present study an incidence of 2.2% of obstructed labour was seen and almost same incidence has been recorded by Datta & Pall (1978) and Sarkar & Paul[2]. Rastogi and Trivedi K[3] gave a higher incidence of 11.78% while Lister[4] noted a lower incidence of 1 in 189 deliveries. The age group incidence in present study was 20-30 years (59.59%) followed by 31-40 years (25.40%) same view is shared by the authors mentioned above. The socio-economic profile of patients in the present study was seen as majority 286 (77.3%) belonging to low S.E. Class of rural areas of Kashmir. Rural preponderance has also been noted by Rastogi and Trevidi[4] and Sarkar & Paul[3]. Rural preponderance has been attributed to : ignorance and illiteracy, low S.E. Status, absence of qualified staff in peripheral hospital. Late referral from peripheral hospitals. The incidence in terms of parity in present study was 196 cases (52.97%) were para 1-4 while 101 cases (29.2[illegible]%) primigravidas. 73 patients were grand multiparas (51). In other comperative studies e.g. by Sarkar & Paul[3] party 1-4 as a major group (57.5%) followed by primigravidas (45.99%) in a series and Ray Chowdery & Sikdar[5] had noted that in primigravidas obstructed labour was more common. Along with age and parity, another correlalting factor was stature. 48.64% of present patients were having a height of 5 ft. 2 inches while 31.35% were below the height of 5 ft. short statured females can have contracted pelvis which gives rise to cephalo-plevic disproportion and hence to obstructed labour. This cause can be prevented by intelligent anticipation during Antenatal period and these patients should be timely shifted to well equipped centers. The most common clinical presentation was due to severe water and electrolyte imbalance in mother giving rise to dehydration and metabolic acidosis. Birth canal infection is almost always established by the time labour has reached the stage of obstruction. Gut distention is another common feature. During the present study CPD was seen in 59.18% cases followed by malpresentation (35.39%). These causes are in close conformity with the finding of Lister[1] and Sarkas of Paul[3]. In a series presented by Roy Chowdery and Sikdar[5], malpresentation has been seen to be the commonest cause. The basic management of obstructed labour is of course urgent delivery to prevent further distension and rupture and to salvage the foets if it is still alive. From this study abdominal delivery by LSCS appears to be the complete answer as justified in present study. The post delivery complications seen were wound sepsis. This was the commonest (31.51%) followed by the post operative shock (22.50%). PPH was seen in 50 patients (16.07%). Rupture uterus as a complication of obstructed labour was seen in 27 cases in this series of 370 cases (7.29%). This incidence is quite significant and it shows the magnitude of late arrivel of cases. The ultimate outcome of this series in terms of maternal and foetal outcome was that there were eight maternal deaths (2.16%) five died of septicemia, 2 due to shock and one patient died becuase of DIC. Less number of maternal deaths reflect the proper management and better case received by the patients admitted with obstructed labour in this study. In foetal outcome there were 14.0 perinatal deaths and an incidence of (37.83%). 84 were still births and 56 Neonatal deaths. In maternal morbidity, the most important long-term sequelae of obstructed labour is VVF. Only 4 cases were reported in present study, because of the following measure taken immediately in hospital i.e. indwelling catheter and destructive operations were less often resorted to, Liste[1] in noted VVF in 21 patients in a series of 32 cases and Sarbar & Paul[3] recorded only 3 cases in a series of 742 cases.

References:

1. Lister U.G. Obstructed labour Journal of obstetrics and Gynaecology Brit. Empire. 1960;72:877.
2. Dutta D.C. and Pal S.K. Obstructed labour. A review of 307 cases. Journal of obstetrics and Gynaecology India. 1987;28:55.
3. Sarkar C.S. & Paul S.K. Obstructed labour a study of 742 cases. Journal of obstetrics and Gynaecology India. 1990;40(5):648.
4. Rastogi S & Trivedi K. Obstructed labour Medicine and Surgery. 1989;29:13.
5. Roy Chowdery N.N. & Sikdar K. Obstructed labour. Journal of obstetrics and Gynaecology India. 1980;30:284.

[illegible]

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end plates of these two vertebrae, more pronounced anterolaterally. MRI (Fig. 2) revealed, decreased intervertebral disc space at D12-L1, with increased signal intensity on T2, and decreased signal intensity on T1 weighted images and questionable desiccation of the disc.

After investigating the patient and taking into consideration his presenting symptoms and positive Widal, a diagnosis of

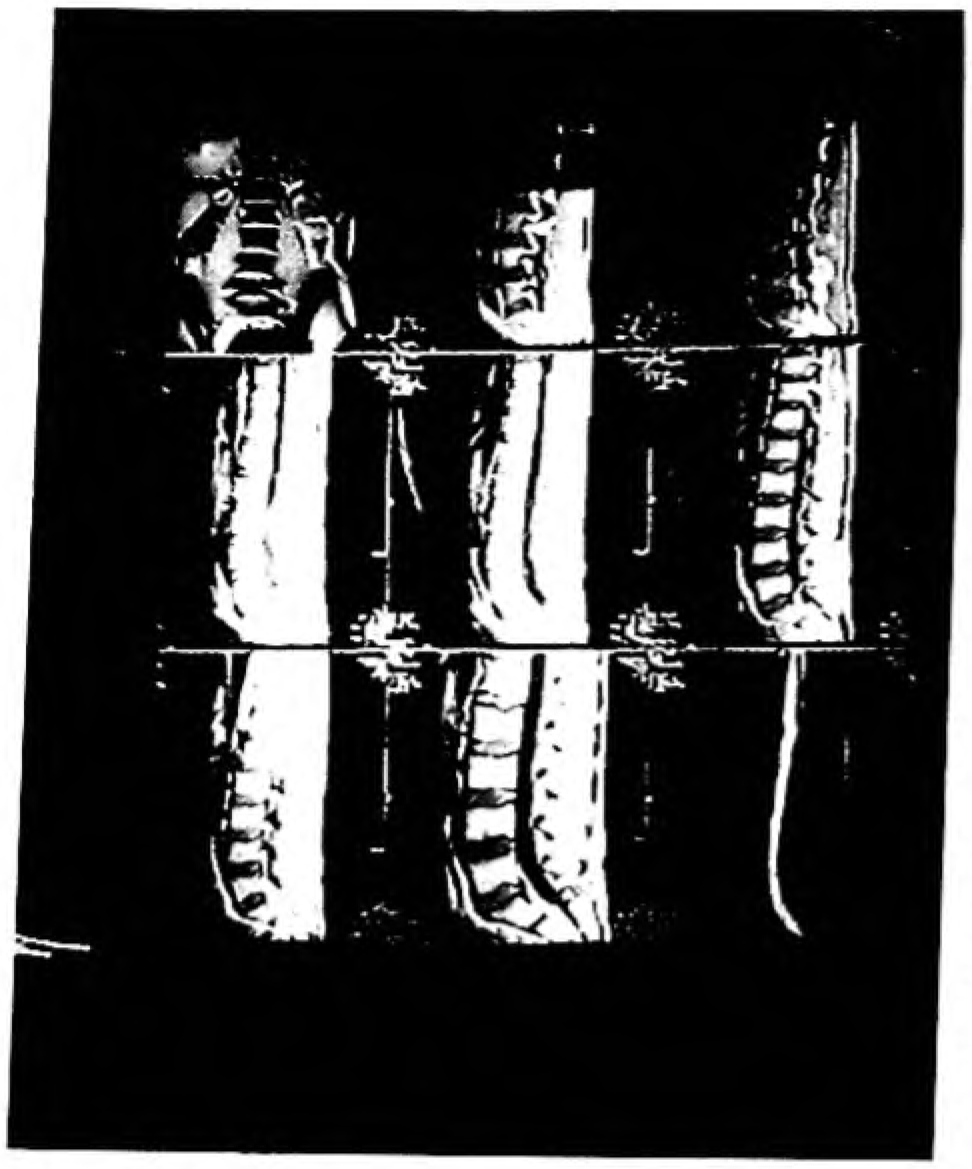

Fig.2: MRI showing disc space reduced at D12-LI with end plate erosion suggested by increased signal intensity on T2, and decreased intensity at T1 weighted images.

no back pain, with improved movements of spine and completely apyrexial. Radiographs did not show any further deterioration.

DISCUSSION:

In reviewing the literature, not many cases of salmonella vertebral osteomyelitis could be found[3-11]. In an endemic area gastrointestinal symptoms usually precede infection by one to twelve weeks[12]. A case of salmonella spondylitis, presenting after 15 months of gastrointestinal symptoms has been reported[13]. In our catchment area, where typhoid fever is endemic, and tuberculosis is very common, it is important to obtain a bacteriological diagnosis, since salmonella osteomyelitis is clinically and radiologically indistinguishable from that due to other organisms. Radiographic findings of vertebral osteomyelitis typically include narrowing of disc space. Most of the cases have been treated non-operatively. A German article describes a 59 year old male veterinary surgeon with a back ache, blood cultures were positive for Salmonella enteriditis, treated conservatively with ciprofloxacin with good results[9].

In conclusion, .one should suspect vertebral osteomyelitis in anyone with fever and back pain .Salmonella infection should be considered if there has been a recent gastro-intestinal disturbance or a positive source of contamination. Although salmonella osteomyelitis is rare, it does occur in developing countries and has to be investigated among the unusual but possible causes of vertebral osteomyelitis. The vertebral bone infection can be treated successfully with proper antibiotics and may require surgical intervention in those whose back pain progresses.

REFRENCES:

1. Murphy JB; Bone and Joint disease in relation to typhoid fever. Surg. Gynaecol. Obstet; 1916,23:1;19-43.
2. Veal JR; McFetridge EM:- paratyphoid osteomyelitis, A report of two cases. JBJS 1934;16:445-50.
3. Ingram R; Reddy P:-salmonella virchow osteomyelitis, A case report. JBJS 70-B,No3, May 1988;440-442.
4. Dolan SA; Everett ED; Harper MC:-Salmonella vertebral OM treated with cefotaxime. Arch. Intern. Med;1987,147.
5. Chamber ST.Wilson AD,Seal DV,Rogers KB:-Paratyphoid fever. J.Infect.10;1985.
6. Doppert E;et al:-Arch.fr.pediatr;47,1990.
7. Orik-Neu C;Mau JS;Cherubin CE, et al:-Bone and joint infection due to salmonella. J.Infect. Dis 1978;138,820-8.
8. Hoffer FA; Strand RA; Gebhardt MC:-percut. biopsy of pyogenic infection in children. J pediatr. Orthop, 8:442-4,1988.
9. Perras B; Kraft B; et al:-Deutsche medizinische 118;1993.
10. Soub HAL; Uwaydah AK; Hussein AH:-vertebral OM in Quatar. British J. Clinical pract, 48,1994.
11. Wozasch CE, Muddowny DS; Isaza JE; Holt RT:-Salmonella vert.OM.A case report. Spine Surgery, Kentucky (Source-Internet).
12. Warren CPM:-Arthritis associated with salmonella infection. Ann. rheum, Dis; 1970;29;483-7.
13. Schmeitzer G;et al:-salmonella spondylitis, an unusual presentation. S Afr.Med J;1971,45;126-8.

CONCOMITANT TUBERCULOUS AND SEPTIC ARTHRITIS OF KNEE JOINT IN A DIABETIC PATIENT

Basharat Hussain Shah M.B.; Tanveer Mir MD, FACP

***Abstract:** A 84 year old Portuguese female presented with right knee pain and swelling of one week duration following a trauma to her right knee. Her past medical history was significant for diabetes. Arthrocentesis revealed straw colored fluid with high cell count and culture grew **staphylococci aureus** resistant to pencillin. In addition the culture also showed acid fast bacilli, later identified as **mycobacterium- tuberculosis.** The synovial biopsy revealed chronic granulomatous inflammation with focal necrosis. The patient was started on intravenous nafcillin and four drug anti-tubercular regimen with resolution of the symptoms over the next few weeks.*

Key Words: arthritis, septic, tuberculosis, diabetic.

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Introduction:

Skeletal tuberculosis is the third most common form of extra-pulmonary tuberculosis following lymphocytic and pleural types. There have been cases reported where the initial diagnosis was made as septic arthritis but later proved as tuberculous arthritis[1]. The concomitant occurrence of septic and tubercular arthritis is extremely rare. The superimposed septic arthritis can change the clinical characteristics of chronic pain with minimal inflammation seen in tuberculous arthritis into acute severe pain with gross inflammation. The diagnosis should always be kept in mind when a patient in whom skeletal tuberculosis is suspected comes with an acute flare up of the disease. Prompt treatment at the initial stage can prevent joint deformity along with patient morbidity.

Case report:

A 84 year old Portuguese female was admitted with complains of right knee pain and swelling for the past one week. There was a history of trauma to the same knee following a fall at home one week back. Patient also reported a draining sinus at the lateral side of the same knee about three weeks ago. There was a straw colored discharge coming from the sinus. Patient past medical history was significant for diabetes mellitus, hypertension, hypothyroidism and atrial fibrillation. Her medications included Levo-thyroxine, Digoxin and multivitamins. Patient had no known drug allergies. Patient had no past or family history of pulmonary tuberculosis. There was no history of cough, weight loss or night sweats. PPD test was negative and patient was not allergic. Patient was also HIV negative.

On examination patient was febrile (102.7°F), pulse of 80 beats per minute and blood pressure of 124/72 mmHg. HEENT was unremarkable. Chest examination revealed bilateral equal air entry and no adventitious sounds. The right knee was swollen, warm and boggy with an obvious effusion. There was restricted active and passive movements on flexion and extension of the right knee. A straw colored discharge was also noted from the sinus on the lateral side of the right knee. (Photograph 1) All other joints were normal. Rest of the physical examination was normal.

Lab. Data on admission revealed white cell count of 8.9 mm^3 with neutrophils of 56%, bands of 19%, lymphocytes of 11%, monocytes of 12% and eosinophils of 2%. The ESR was 86 mmhr (predicted 0-30 mmhr). The C-reactive protein(CRP) was 18.5ug/ml (predicted 0-1ug/ml). Rest of the laboratory results were normal. **Joint fluid aspiration** revealed straw colored fluid and WBC count of 60,000 mm^3 with neutrophils of 82%, lymphocytes of 12%, monocytes of 6% and synovial lining cells of 2 per 100 WBC. The **joint fluid smear** did not show AFB or any fungal elements.

Joint fluid culture grew staphylococci aures resistant to pencillin. The culture also grew acid fast bacilli, later identified as mycobacterium tuberculosis. There was no fungal growth seen. No crystals were seen in the joint fluid. **Arthroscopy** of the right knee was done and biopsy was taken from synovial membrane and bone which showed chronic granulomatous inflammation along with foci of necrosis. **Chest radiograph** was normal. The **radiograph**

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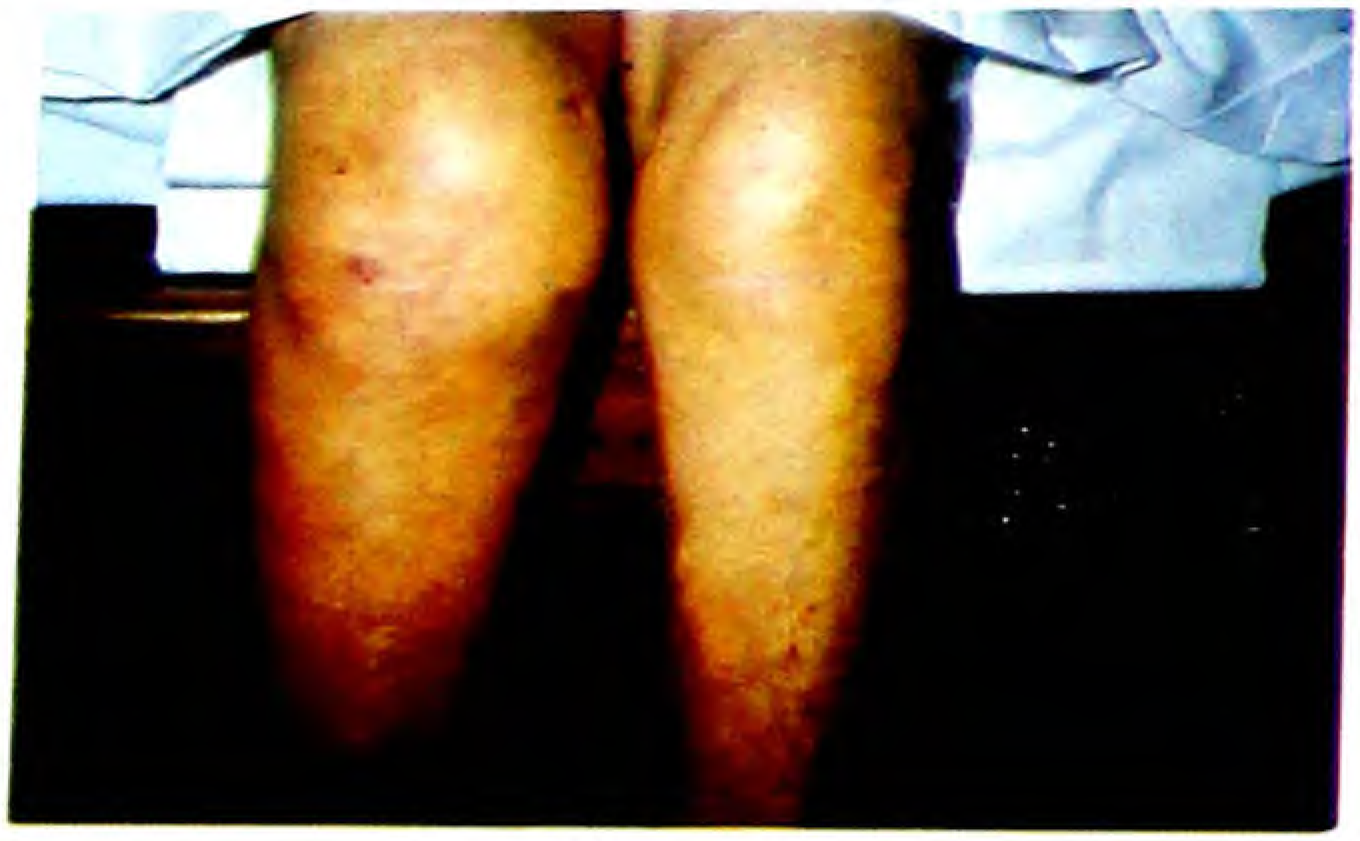

Figure. 1

of right knee showed periarticular osteopenia along with joint space narrowing and marginal erosions of lower end of femur and upper end of tibia. There was no evidence of fracture or dislocation. The patient was started on intravenous naficillin along with rifampicin, isoniazid, ethambutol and pyrizinamide. There was a gradual resolution of the sinus and swelling of the knee over the next 4-5 weeks. The patient was then followed on an out patient basis.

Discussion:

Skeletal tuberculosis is a rare disease usually caused by the spread of mycobacterium tuberculosis reaching via the blood stream to either the synovium or the bone. It has been found to occur in about 1% to 3% of all patients with tuberculosis and makes up to 10% of all extrapulmonary forms reported. However in recent years there has been a resurgence of musculoskeletal tuberculosis in the United States. This has been attributed to the AIDS epidemic and the increased travel and immigration from the underdeveloped countries[2]. The joints known to be involved in tuberculosis are spine (50%), pelvic (12%), hip and femur (10%), knee and tibia (10%), ribs (5%), ankle and shoulder (2%), elbow and wrist (2%) and multiple sites (3%). There is a paucity of data in the recent literature concerning tuberculosis of the knee[3]. Superimposed pyogenic arthritis can occur but is extremely rare and can change the clinical presentation making diagnosis difficult. The tuberculous arthritis is usually monoarticular as in septic arthritis but can present as polyarticular arthritis as well. Only 50% of the patients with skeletal tuberculosis demonstrate clinical or radiologic evidence of pulmonary disease. The chest radiograph may show old or active pulmonary tuberculosis in 50% of the cases[4]. There is usually a delay in the diagnosis of skeletal tuberculosis because patients have mild chronic pain and minimal signs of inflammation. The onset is usually insidious. Unlike pyogenic arthritis, tuberculous arthritis is usually not associated with spiking fever or generalized malaise, although systemic symptoms such as low grade fever, weight loss, night sweats are sometimes present[5]. The tuberculin skin test remains one of the most valuable diagnostic tools and is often positive in 90% to 100% of non-anergic patients, however, it is not completely reliable[6]. However, a negative skin test does not absolutely rule out tuberculosis. Patients with AIDS usually have a negative skin test. The erythrocyte sedimentation rate is usually elevated. The white cell count is usually normal. In skeletal tuberculosis intermittent effusions are common and there is a precipitation of fibrin in it giving rise to ***rice bodies.***

The diagnosis of skeletal tuberculosis is based on the following four features (***Ellis et-al***)[7] which include absence of evidence for alternate infective arthritic processes, absence of evidence for active non-infective arthritis-including gout, rheumatoid arthritis and connective tissue diseases, presence of a swollen, cold joint, accompanied by a history of at least 2 weeks of pain together with presence of ankylosis and/or swelling and/or sinus and positive culture from joint fluid or synovium for Mycobacterium tuberculosis or caseating granulomas on tissue biopsy or eosinophilic staining epithelioid cells, langhan giant cells and lymphocyctic aggregates highly suggestive of tuberculosis. The identification of the mycobacterium tuberculosis organism on the smear or in culture is the most reliable method of establishing the diagnosis[3]. It is rare to identify the acid fast organism on the smear but the cultures can sometimes help in diagnosis which usually take 4-6 weeks. About one-fifth of the patients with tuberculous arthritis will have a positive synovial fluid smear for tubercle bacilli[7]. The synovial fluid white cell count is usually 10,000 to 20,000 cells/mm^3 but it varies widely. The classical synovial pathology revealing chronic granulomatous inflammation with epithelioid histiocytes and multinucleated giant cells including langhans type giant cells with focal areas of necrosis establish the diagnosis in 90% of the cases while histologic examination along with culture establish the diagnosis in 95% of the cases because granulomatous inflammation alone is seen in other conditions such as atypical mycobacteria, certain fungi, brucella species, sarcoidosis, crystal associated arthritis and foreign body reactions[8].

Berney et al showed that synovial fluid smear stained for acid fast organism will be positive in only 27% of the tuberculous joints[9]. Simple aspiration of the joint is much less likely to lead to a definitive diagnosis. Stains to identify acid fast bacilli reliably require the presence of at least 10^4 acid fast organism per milliliter of specimen, but cultures are more sensitive and can reliably identify mycobacteria in a concentration of 10^3 organisms per milliliter of specimen[10]. Soft tissue swelling along with sinuses and cold abscesses are common in tuberculous arthritis. The radiographic findings can show patchy osteopenia, marginal erosions and joint space narrowing. In more severe cases osteolytic lesions with sclerotic margins are seen in the metaphysis which may extend into epiphysis[11]. A triad of radiological findings with ***Phemisters triad*** is characteristic of tuberculous arthritis and includes intra articular osteoporosis, peripherally located osseous erosions and gradual narrowing of the interosseous space[12].

Marks and McHenry divided tubercular arthritis into four classes based into severity of the disease[5].

Class I includes infection of the synovium with little or no osseous involvement.

Class II includes infection of the synovium and bone ; the joint space is preserved.

Class III includes extensive osseous and synovium involvement and progressive lost of the joint space.

Class IV is tuberculous arthritis of any stage but with a ***super imposed pyogenic infection.***

FEATURES OF OUR CASE:

Our patient had an acute onset with short durations of symptoms. The patient did not have clinical or radiological evidence of pulmonary tuberculosis. Only 50% of skeletal tuberculosis have such evidence. The skin test was negative which however is not completely reliable. The white cell count was significantly raised with left shift because of concomitant pyogenic arthritis. The culture grew both staphylococcal aureus as well as mycobacterium tuberculosis. The synovial pathology revealed chronic granulomatous inflammation with focal necrosis. The patient fitted into the class IV of Marks and McHenry classification It has been suspected that trauma may play a role in the pathogenesis of bone and joint tuberculosis[13] as seen in our patient. The presence of underlying debilitating condition such as diabetes mellitis as in our patient can predispose to tuberculous arthritis with superimposed septic arthritis[14].

In summary tuberculous arthritis is not a difficult diagnosis when the classic Phesmisters triad is present along with relevant clinical symptoms. However, when the presentation is unusual as in our case, other things to keep in mind are bacterial or fungal infection or monoarticular rheumatoid arthritis. Hence it is important that routine synovial fluid should be send for both bacterial and tuberculous culture.

REFERENCES:

1. Ellis EM, Ramahi El M, Al- Dalaan NA : Tuberculosis of peripheral joints : a delimma in the diagnosis. *Tubercle and Lung Disease 1993;74:399-404.*
2. McKenna MT, McCray E, Onorato I : The epidemiology of tuberculosis among foreign born persons in the United States. *N Engl J Med. 1995;332:1071-76.*
3. Furia PJ, Box GG, Lintner MD: Tuberculous arthritis of the knee presenting as a meniscal tear. *Am. J. of Ortho. 1996;25:138-42.*
4. Weir MR, Thorton GF : Extrapulmonary tuberculosis : Experience of a community hospital and review of the literature. *Am J Med. 1985;79:467-78.*
5. Marks KE, McHenry MC : Nonspinal tuberculosis. Ini Evarts CM, ed. *Surgery of the musculoskeletal system. New York: Churchill Living Stone; 1990:4557-72.*
6. Jokl P, Goodman AH : Skeltal tuberculosis : diagnosis of three unusual cases. *Clin Orthop Relat Res. 1972;85:109-14.*
7. Wallace R, Cohen SA : Tuberculous Arthritis : A report of two cases with review of biopsy and synovial fluid. *American Journal of Medicine 1976;61:277-82.*
8. Kostman RJ, Rush P, Reginato JA : Granulomatous tophaceous gout mimicking tuberculous tenosynovitis : Report of two cases. *Clinical Infectious Diseases 1995;21:217-9.*
9. Berney S, Goldstein M, Biskho F : Clinical and diagnostic features of tuberculous arthritis. *Am. J. Med. 1972;53:36-42.*
10. Rooney JJr. Crocco JA, Lyons HA : Further observation on tuberculin reactions in active tuberculosis. *Am. J. Med. 1976;60:517-22.*
11. Aguirre M, Bago J, Martin N : Tuberculosis of the knee - Surgical or Conservative treatment? *Acta Ortho Bel 1989;55:22-25.*
12. Resnick D : *Diagnosis of Bone and Joint Disorders. 2th Ed. Philadelphia , PA : WB Saunders;1984;4:2661-86.*
13. Wright T, Sundram M, McDonald D : Tuberculous osteomyelitis and arthritis. *Orthopedics 1996;19:700-702.*
14. Davidson TP, Horowitz I : Skeltal tuberculosis - A review with patient presentation and discussion. *Am. J. of Med. 1970;48:77-84.*

HYDATID CYST IN THE NECK -AN UNUSUAL PRESENTATION

Rafiq Ahmad Pampori; Asef Wani; Manzoor Latoo; Arshad Hakeem

Abstract: *Hydatid cyst in the cervicofacial region is rare. We present an unusual case of a hydatid cyst found in the neck under the sternomastoid muscle going deep down to the superior mediastinum. The location of the lesion, diagnostic problems and therapeutic approach are discussed. Review of literature is also presented.*

Key words: *Cyst, Hydatid, Neck.*

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Introduction

Echinococcal parasites are members of the order Cestoda (flatworms) and the family Taenia. The propensity for these organisms to cause infection in pastoral and other rural setting is best explained by their life cycle. the adult form of echinococcus is a 5mm long hermaphroditic tapeworm which infests the small intestine of carnivorous animals, such as cows, sheep, buffaloes, zebra, moose and caribou. In the duodenum, enzymatic digestion of the egg-shell releases embryonic forms of the organism, which then pass through the mucosa of the small intestine and enter the portal circulation. Once filtered by the liver or the lung the embryo transforms into a microscopic larval stage, the protoscolex or scolex, which is capable of multiplying asexually within the affected organ. "When the host animal dies or is slaughtered, ingestion of organs infested with larva completes the life cycle, and the larva mature into adult parasites within the small intestine of the carnivore.

Man is an accidental, intermediate host in the echinococcal life cycle, as infection of human beings represents a terminal event for the parasite. Typically, man is exposed to the organism by ingestion of contaminated vegetables, or meat, which cannot be washed or scrubbed free of eggs. Infection may also occur when playing with dogs harbouring the tapeworm, as eggs cling to their fur. This is the most common means by which children acquire the disease. The larval form of echinococcus can invade any organ system, and the distribution of infection is limited only by blood flow and filtration. Thus, hydatid cyst can occur in the liver, spleen, lungs, brain, muscle, and even bone. In humans, approximately 65% to 75% of hydatid cysts occur in the liver, 25% are found in the lungs, and 5% to 10% distribute along the peripheral arterial system.

Case Report:

A 15 year old female presented with a slowly progressing non tender swelling neck, (L) side, of eight months duration. The swelling was not associated with symptoms like pain, dysphagia, odynophagia, hoarseness of voice or weight loss etc. There was no other swelling in any other part of the body. GPE was normal, on local examination there was a non tender swelling extending above from the middle of the neck about 3 fingers below the mandible, medially from medial border of the sternomastoid upto the lateral border of sternomastoid laterally. The swelling was smooth in surface and firm in consistency. Upper, medial and lateral edges were palpable whereas the lower edge was not palpable as it was going under the clavicle. The swelling did not move with deglutition or protrusion of tongue. Skin overlying the swelling was free and it was not a pulsatile swelling.

Routine haemogram, x-ray chest and USG abdomen was within normal limits. On FNAC clear fluid was aspirated nd Pathologist reported cystic swelling neck.

Surgery: The swelling was exposed using a horizontal neck crease incision. Shinning white cyst wall was found, the cyst was going deep under the sternomastoid even below the clavicle into the superior mediastinum. A wide cryoprobe was used to pull the cyst in order to get it out in toto. A typical hydatid cyst was found which was sent for HPE which confirmed the diagnosis. Post-operative period was uneventful.

Serology: IgG serology for hydatid cyst was advised postoperatively as there was no suspicion of hydatid disease preoperatively. It was reported positive with the reading of 1.4.

Discussion

The diagnosis of echinococcosis (echinococcus granulosus) in an atypical location can be difficult to make and frequently can only be established by histological examination of affected tissues. Surgical excision and fine needle aspiration biopsy usually lead to the diagnosis. Since puncture of these cysts may lead to an anaphylactic reaction

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LAPAROSCOPIC TREATMENT OF HYDATID CYST OF LIVER.

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ABSTRACT: Laparoscopic partial cystopericystectomy of the Hydatid Cyst of liver is based on safe and sound surgical principles. The benefits of laparoscopic surgery should be extended to the patients suffering from this benign yet common condition.The method is less traumatic and requires a shorter hospital stay with a faster recovery.

KEY WORDS: Ecchinococcosis. Hydatid cyst liver, Laparoscopy, Partial cystopericystectomy.

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INTRODUCTION

Echinococcosis is the most frequent cause of liver cysts in the world. The problem being endemic in the sheep rearing regions. The prevalence of a large population of stray dogs increases the incidence.The most common form is due to Echinococcus granulosus, a 6 mm dog tapeworm(1).

Hydatid cysts of the liver are characterized by an indolent yet unremitting growth in the majority of infected patients, with the potential for cyst metastases to the peritoneal cavity or lungs.Hydatid cysts show slow growth rates and rarely cause systemic symptoms. Approximately 75% of cases present as an asymptomatic abdominal mass or are picked up on an abdominal ultrasonograph y ordered for some other complaint(2).

These patients usually present with an abdominal swelling , abdominal distention , subcostal heaviness, jaundice and fever (infected) etc.

The conventional operative procedures like enucleation, cystectomy, evacuation, marsupialisation , etc., involve a significant morbidity especially in terms of wound infections, etc(3).

Recently successful attempts have been reported for the management of hydatid disease of the liver by laparoscopic methods. The following case report illustrates the feasibility of this technique.

CASE REPORT

A 9 year old girl presented with a swelling in the right hypochondrium with a constant dull ache in the region of one year duration. There was no other significant complaint. The girl was from a village where sheep rearing was the main occupation and the population of stray dogs was significant.

Routine investigations revealed a normal haemogram, kidney functions and liver function tests. Abdominal ultrasonography revealed a single unilocular cyst on the anterior aspect of the right lobe of the liver.The cyst measured 8 x 9 cms.Rest of the liver and abdominal viscera were normal. Casoni's test and ELISA for hydatid disease were positive. CT scan of the abdomen was performed for noting the exact site and dimensions of the cyst. It was decided to manage this case laparoscopically, young age of the patient being the prime factor.

OPERATIVE PROCEDURE

The patient , under general anaesthesia , was placed supine on the table with the surgeon and the camera assistant standing on the left. The second assistant was positioned on the right side. Video monitors were placed on both sides .Pneumoperitoneum was created by Veress needle puncture at the umbilicus. The pressure was set at 12 mmHg. A 10 mm port for the telescope was inserted at the umbilicus. After examining the peritoneal cavity and localizing the cystic area (bumpiness), three more ports were inserted .One (5 mm) to the right of the umbilicus,second (10 mm) to the left of the umbilicus and the third (5 mm) to the left of the xiphoid process. Another (10 mm) port was inserted just below the xiphoid process.

Gauze pieces soaked in 0.5% cetrimide solution were inserted around the operative site, through the 10 mm xiphoid port . Grasper was introduced through the left umbilical port to stabilize the liver .The cyst was punctured with a long laparoscopic needle connected to high vacuum suction ,through the xiphoid port. Another sucker was introduced through the right mm umbilical port to avoid accidental spillage. Around 100 ml of cyst fluid ,clear in colour, was aspirated carefully and 50 ml of 0.5% cetrimide solution was put in via the same needle. The procedure as repeated four times , at 5 min intervals. After this 250 ml of the cyst fluid was aspirated . The needle was withdrawn while still connected to suction to prevent

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6. Khuroo M S, Dar M Y, Yattoo G N , Zargar S A, Javaid G, Khan B A, Boda M I . Percutaneous drainage versus albendazole therapy in hepatic hydatidosis:A prospective randomized study. Gasteroenterology 1993; 104 : 1452-1459.
7. Simonetti G, Profili S , et al . Percutaneous treatment of hepatic cysts by aspiration and sclerotherapy. Cardiovas Intervent Radiol 1993;16:81.
8. Metin Ertem , Cihan Uras, Tayfun Karahasanoglu, Sabri Erguney, Kemal Alemdaroglu. Dig Surg 1998; 15 :333-336.
9. Sayek I, Cakmakci M . Surg Laparosc Endosc 1996 Feb; 6(1) : 16-21.
10. Bickel A, Daud G, Urbach D, Lefler E, Barasch E F, Eitan A. J S L S 2001 Jul-Sep; 5(3) :237-40.
11. Saglam A Surg Endosc 1995 Dec ; 9(12) : 1304-5.
12. Alper A, Emre A, Hazar H, Ozden I, Bilge O, Acarli K, Ariogul O . Laparoscopic surgery of hepatic hydatid disease: Initial reports and early follow-up of 16 patients. World J Surg 1995 ;19 :725-728.
13. Monpean A L, Paricio P P, Campos R, Ayllos J G. Laparoscopic treatment of a liver hydatid cyst. Br J Surg 1993; 80:907-908.

APPLICATION OF MECHANICAL VENTILATION AND BRONCHOSCOPY IN THE MANAGEMENT OF BRONCHOPLEURAL FISTULA

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Bronchopleural fistula (BPF) is an aberrant communication between bronchial tree and pleural space through which inspired gas exits the lungs. It is a formidable therapeutic and management challenge especially in patients on mechanical ventilation, since very little information is available on its natural history and prognosis. In emphyema, which is a medical emergency, adequate early pleural drainage is crucial because if the fluid is not drained exteriorly with chest tube, it is likely to drain internally into the lungs producing an overwhelming pneumonia. BPF is involved not only in the drainage of gas from the alveoli in the vicinity of the fistula but also from remote alveoli by pressure gradients created by it. It leads to decreased utilization of inspired oxygen before it esscapes through fistula and is also an efficent mechanism of carbon dioxide removal.

Approximately two-thirds of BPF cases complicate lung surgery i.e. pneumonectomy (5-20%), lobectomy (1-5%), segmentectomy (0.5%) and wedge resection (0.5%). Remaining one-thrid of BPF cases are related to pleuropulmonary tuberculosis, necrotising pneumonia, lung cancer, suppurative lung diseases, ventilator therapy in ARDS, chest trauma (blund or pnetrating injury, thoracocentesis, pleural biopsy, chest tubes, CVP lines) and alveolar rupture which may be spontaneous or related to treatment of ARDS. In addition, BPF can complicate histoplasma capsulatum associated lymphocytic empyema as well as pancreating pleural effusion. However in developing countries including India tuberculosis continues to be etiologically very important in BPF.

Clinical presentaiton of BPF ranges from incidental discovery on chest x-ray to life-threatening tension pneumothorax, however the most common presentation is a chronic wasting illness that is fatal in months to years if untreated. BPF should be suspected when a patient with pleural fluid collection expectorates more sputum than expected from the associated pulmonary disease or when a patient produces large amounts of sputum only when lying in one position. In acute presentation, large quantity of infected material can flood the airways of both ipsilateral and contralateral lung producing acute respiratory compromise. Early post-surgery BPF can have dramatic presentation because the mediastinum is mobile and predisposes to contralateral shift theraby compromising the remaining lung function. In contrast, late post-surgery BPF arises in fibrotic space thus impeding the development of tension pneumothorax due to fixed mediastinum.

Diagnosis of BPF is usually not difficult and is evident from clinical events. Upright chest x-ray showing an air-fluid level particularly if the level fluctuates in serial films is highly suggestive of BPF. Sometimes it is difficult to differentiate from chest x-ray whether the air-fluid level is in lung parenchyma or pleural space, a problem that is solved by ultrasound and CT scan. Thin section CT scan has been reported to be superior to standard CT especially in the diagnosis of peripheral fistulas. Bronchography documents the bronchopleural communication. Other highly useful investigations for the diagnosis of BPF include xenon-ventilation nuclear scintigraphy, thoracoscopy and intrapleural injection of methylene blue followed by bronchoscopy.

Tubercular BPF is uncommon nowadays becuase of effective antitubercular drugs (ATDs) and is usually seen in association with inadequate treatment, cavity rupture during active disease and old healed pulmonary tuberculosis with fibrothorax. The two important dangers of tubercular BPF are the propensity of mycobacteria in pleural space to become ATD resistant and occasional bacterial superinfection of pleural space, which may produce fulminant pneumonia. The incidence of this type of fistula is high in India as compared to European countries and USA, however data for comparison is not known. For management conservative approach that includes ATDs and chest tube drainage is preferred and may be sufficient in one-third of patients. Before a definitive surgery is undertaken patient should ideally receive ATD for three to four months or until sputum is AFB negative. Drainage of pleural space is crucial first step for all patients to limit endobronchial contamination. By the time BPF develpps the disease is so advanced as to preclude satisfactory re-expansion of lung thereby limiting surgical options to open thoracotomy or multiple stage thoracoplasty. Extensive disease may need decortication or pleuropneumonectomy. For surgically unift patients, bronchoscopy related treatment

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6. Khuroo M S, Dar M Y, Yattoo G N , Zargar S A, Javaid G, Khan B A, Boda M I . Percutaneous drainage versus albendazole therapy in hepatic hydatidosis:A prospective randomized study. Gasteroenterology 1993; 104 : 1452-1459.
7. Simonetti G, Profili S , et al . Percutaneous treatment of hepatic cysts by aspiration and sclerotherapy. Cardiovas Intervent Radiol 1993;16:81.
8. Metin Ertem , Cihan Uras, Tayfun Karahasanoglu, Sabri Erguney, Kemal Alemdaroglu. Dig Surg 1998; 15 :333-336.
9. Sayek I, Cakmakci M . Surg Laparosc Endosc 1996 Feb, 6(1) : 16-21.
10. Bickel A, Daud G, Urbach D, Lefler E, Barasch E F, Eitan A J S L S 2001 Jul-Sep; 5(3) :237-40.
11. Saglam A Surg Endosc 1995 Dec ; 9(12) : 1304-5.
12. Alper A, Emre A, Hazar H, Ozden I, Bilge O, Acarli K, Ariogul O . Laparoscopic surgery of hepatic hydatid disease: Initial reports and early follow-up of 16 patients World J Surg 1995 ;19 :725-728.
13. Monpean A L, Paricio P P, Campos R, Ayllos J G Laparoscopic treatment of a liver hydatid cyst. Br J Surg 1993; 80:907-908.

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to effect fistula closure is preferred. Alternatively, long term chest tube may be advised.

Prognosis depends primarily on nature and extent of underlying lung disease, prevence or absence of pleural space infection and associated comorbid condition like poorly controlled diabetes, chronic renal failure, decompensated liver diseaes. immunodeficiency etc. The size of the air-leak is an independent vital prognostic factor - leaks exceeding 500 ml/breath are almost always fatal where as leaks less than that have significantly lower mortality rates. Similarly, in chest trauma patients, leaks appearing within the first 24 hours of injury have a relatively better prognosis (45% mortality) as against leaks appearing after that (90% mortality). Overall patient survival depends on high index of suspicion, early diagnosis and aggressive surgery.

Management issues:

Best management of post-surgery BPF is prevention, including meticulous surgery coupled with liberal prophylactic use of pedicled muscle flaps for patients at increased risk. Equally important is the treatment of underlying lung disease, appropriate antibiotics and good nutritional status of the patient. Initial treatment includes effective drainage to prevent flooding of brocnhial tree with pus. Increased flow through fistula is an important factor that delays healing and in general flow is decreased when the side with fistual is placed in dependent position.

Intercostal chest tube drainage (CTD) has far more important role than merely of a passive conduit in BPF management and can be both useful as well as detrimental to the patient. It not only drains pleural air due to fistula but also decreases the air leak thereby promoting fistula repair. Efficient drainage is very crucial since air leakes may be as high as 16 liters per minute. Small diameter chest tube can lead to lung collapse and tension pneumothorax in the seeting of mobile mediastinum. Large diameter chest tube is particularly indicated in high flow BPF and infected pleural space. Amount of suction applied via ICTD is individualised since the requirements may very from no suction to intermediate suction (10-15 cm of water) to high suction($\geq$25 cm of warer). The negative effects of ICTD include loss of tidal volume, altered PaO_2 and $PaCO_2$ and interference with ventilator cycling.

Mechanical ventilation and BPF:

In case of inoperable BPF all techniques of ventilation (and pleural drainage system) should be tried, since there is no consensus on the effects of ventilatory adjustments on duraiton and healing of air leak. Management of large BPF of a ventilated patient is difficult as achievement of adequate ventilation invariably impairs fistula repair. The amount of air leak is inversely related to patient survival since leak via fistula delays its healing. The main threat is hypoxia and not hypercarbia because the air that leaves BPF has CO_2 level comparable to that in mixed expired air.

The targets of conventional ventilation (CV) in BPF management include lowest effective tidal volume and PEEP together with lowest possible number of mechanical breaths and decreased inspiratory time. These goals are met more accurately with SIMV rather than ACMV mode. Pressure control ventilation (PCV) has been successfully used in treating BPF that develops in the setting of polytrauma related ARDS. By allowing lower inspiratory airway pressures PCV promotes fistula closure.

High frequency jet ventilation (HFJV) is a United States FDA approved method for ventilating BPF patients, however in a given patient it may not be the optimal choice of ventilation. It is generally superior to CV in maintaining PaO2 and PaCO2. It is particularly recommended for managing tracheal or proximal bronchial fistulas with normal lung parenchyma. HFJV decreases flow through BPF, an effect seen more consistently in infants and young children than in adults and is usually accompanied by a drop in PaO2.

In ARDS, BPF typically occurs after one to two weeks of illness and is associated with poor prognosis. The fistula flow increases significantly with each increment of PEEP irrespective of the method of ventilation (CV vs HFJV). Therefore HFJV is justified in ARDS only if CV fails in managing associated BPF. In ARDS related BPF, the HFJV does not provide adequate gas exchange and the leak itself is not affected because of peripheral location of fistula and decreased lung compliance.

Other ventilatory options that are helpful whether using CV or HFJV in BPF management include

1. Selective intubation and CV of unaffected lung.
2. Independent lung ventilation, wherein two ventilators
   are used - HFJV applied to BPF containing lung and CV to normal lung.
3. Ultrahigh frequency jet ventilation applied to BPF containing lung and CV to normal lung.

Role of bronchoscopy:

Fiberoptic bronchoscopy (FOB) has both diagnostic and therapeutic role in BPF. It can be used to directly visualize and treat fistulas particularly the proximal ones and to assess the length of the bronchial stump harbouring the fistula. Being a relatively non-invasive treatment option bronchoscopy is highly valuable in poor surgical candidates. Distal BPFs not directly visualized by bronchoscope require passage of an occluding ballon following which a decrease in air leak localizes the fistula containing bronchial segment. For directly visualized proximal BPFs sealent is applied through a catheter in the working channel of bronchoscope whereas for distal BPFs multiple lumen Swan-Ganz catheter is used both to localize the fistula and to pass the occluding material of choice.

Other than balloon occlusion technique, the various materials that can be passed through bronchoscope to effect the closure of BPF include cyanoacrylate-based agents, fibrin, silver nitrate, tetracycline, gelfoam and lead-shots. However, these materials are not useful in large tracheal or proximal bronchial ruptures and multiple distal parenchymal defects. Immediately these agents seal the leak by acting as a plug and subsequently close the fistula by inducing fibrosis and mucosal proliferation.

Recent advances:

1. *Sclerotherapy:*

   It is carried out by bronchoscopic submucosal injection of polidocanal around BPF. It is technically easy and highly successful in treating fistulas complicating pleuropneumonectomy for lung cancer. It is safe, simple and inexpensive and can be prescribed as the first therapeutic step in BPF management.

2. *Nd-YAG laser;*

   This is an attractive endobronchial treatment option for small (<2mm) proximal BPFs. Laser beam is directed through bronchoscope to bronchial mucosa surrounding the fistula. This procedure has about 80 percent success rate in closing fistulas which have no tumour or infection at the bronchial stump.

References:

1. Cerfolio RJ. The incidence, etiology and prevention of post-resectional bronchopleural fistula. Seminars in thoracic and cardiovascular surgery 2001;13(1):3-7.
2. Varoli F, Roviaro G et al. endoscopic treatment of bronchopleural fistula. Annals of thoracic surgery 1998;65(3):807-9.
3. Carvalho P et al. Management of bronchopleural fistula with a variable resistance valve and a single ventilator. Chest 1997;111(5):1452-4.
4. Michael H. Baumann, Steven A. Sahn. Medical management and therapy of bronchopleural fistula in mechanically ventilated patients. Chest 1990;97:721-28.
5. Pierson DJ, Hoston CA, Bates PW. Persistent bronchopleural air leak during mechanical ventilation: a review of 39 cases. Chest 1986;90:321-23.
6. Powner DJ, Grenvik A. Ventilatory management of life-threatening bronchopleural fistula. Crit Care Med 1981;9:54-8.
7. Bishop MJ, Benson MS, Pierson DJ. Carbon dioxide excretion via bronchopleural fistula in adult respiratory distress syndrome. Chest 1987;91:400-2.
8. Roth MD et al. Gas flow through a bronchopleural fistula: measuring effects of high frequency jet ventilation and chest tube suction. Chest 1988;92:210-13.
9. Lan R, Lee C, Tsai Y, Wang W, Chang C. Fibreoptic bronchial blockade in small bronchopleural fistula. Chest 1987;92:944-46.
10. Westcott JL, Volpe JP. Peripheral bronchopleural fistula: CT evaluation in 20 patients of pneumonia, empyema and post-operative air leak. Radiology 1995;196:175-81.

HEMORRHOIDAL DISEASE

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Hemorrhoidal disease is quite a common problem in our valley probably secondary to low fibre diet. Hemorrhoid, from the patient's perspective, has always signified a variety of anal complaints varying from minor itching to acute disabling pain. In the Bible, the Old Testament of 1 Samuel, chapter 5 and 6 describes the Philistines after taking the Ark of the Covenant from the Israelis as being smitten by God with aphelim or techorim. Both words are believed by scholars to relate to hemorrhoids[1,2]. The disease has plagued mankind throughout recorded history[3]. Maimonides was the first to describe the treatment for this disease in the form of soothing medications, ointments and even suppositories [4].

The hemorrhoidal tissue, in humans, is thought to contribute to anal continence by forming a spongy bolster, which cushions the anal canal and prevents damage to sphincter mechanism during defecation[1]. Hemorrhoids are cushions of vascular tissue found in the anal canal[3]. Hemorrhoidal tissue is present at birth and in non-pathological conditions.

Microscopically, this tissue contains vascular structures whose walls do not contain muscle. Thus, hemorrhoids are not veins (which have muscular walls) but are sinusoids [5]. Recent studies have also demonstrated that hemorrhoidal bleeding is arterial and not venous. When these sinusoids are injured (disrupted), hemorrhage occurs from presinusoidal arterioles. The arterial nature of the bleeding explains why hemorrhoidal hemorrhage is bright red and has an arterial PH [6]. During prolapse this region is exposed to atmospheric pressure, and the pressure gradient causes bleeding that is characteristically arterial in type [7].

PATHOPHYSIOLOGY:

The typical Kashmiri low fibre diet may explain the high prevalence of constipation, straining and hemorrhoidal symptoms [8]. The supporting muscularis submucosa leads to prolapse of the hemorrhoidal tissue secondary to weakening and aging. The microscopic evidence is that anal supporting tissue deteriorates by third decade of life [9]. Also, several authors report that patients with hemorrhoids have increased activity of the internal anal sphincter [10,11]. All these conditions contribute toward stretching and slippage of the hemorrhoidal tissue. The overlying skin or mucosa is stretched and additional fibrous and sinusoidal tissue develops. The extra tissue tends to move caudally toward the anal-verge making it susceptible to injury [12]. Hemorrhoids lying in the upper two-thirds of the anal canal are covered by columnar cell epithelium and are classified as "Internal" whileas hemorrhoids in the lower third are covered by skin and are termed as "External".

TREATMENT:

Can be non-operative or operative. Non-operative techniques include sitz baths, dietary modifications and topical medications. Operative techniques include tissue fixation, major tissue excision, or physiological alternations of the anal canal like Lord's procedure or lateral internal sphincterotomy.

TREATMENT OF INTERNAL HEMORRHOIDS BY DEGREE OF PROLAPSE: [3,13]

Severity	Treatment
First Degree (No Prolapse)	Dietary Infrared coagulation Banding. Sclerotherapy
Second Degree (Spontaneously reducible)	Dietary plus Banding Infrared coagulation Sclerotherapy
Third Degree (manual reduction necessary)	Dietary plus Banding Infrared coagulation Sclerotherapy Excisional hemorrhoidectomy
Fourth Degree (Irreducible)	Excisional hemorrhoidectomy Rarely, multiple rubber band ligation
Acutely prolapsed and thrombosed	Emergency hemorrhoidectomy

In addition, the other modalities of treatment which may be used for internal hemorrhoids are: -

Electrocautery: which uses bipolar and direct current device but not with much encouraging results [14,15]

Dilation: described by Lord in 1968 & 1973 [16] in which the procedure entails careful but firm dilatation of the anal canal. The treatment is based on the premise that increased anal pressure contributes to hemorrhoid symptoms [17]

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Internal anal Sphincterotomy: It is a more controlled technique to lower anal pressure but it does not address associated tags or external hemorrhoids [18]. Although, sphincterotomy may be reasonable in the surgical treatment of hemorrhoids with concomitant anal fissure, neither other authors nor we recommend its use as the sole treatment of isolated hemorrhoid disease [1].

Cryotherapy: It is based on rapid freezing and thawing of tissue which theoretically causes analgesia and tissue destruction. Cold liquid nitrogen is delivered for freezing the tissue though a cryoprobe. However, the results are not encouraging because of significant pain, tissue destruction and profuse foul discharge [1, 14, 19, 20].

EXTERNAL HEMORRHOIDS:

The effort or straining may invite thrombosis of an external hemorrhoid. The tissue around these clots swell causing moderate to serve pain. If not treated in 2-4 weeks, the clot in the thrombosed vessels will either spontaneously drain through the thinned overlying skin or be gradually resorbed. Redundant anal skin, which usually remains, needs no treatment. However, patient concern may invite excision under local anaesthesia.

For hemorrhoids with mild symptoms patient needs reassurance, stool bulking agents and pain killers if patient presents early, the procedure of choice is excision. The procedure can be done under local anaesthesia and should remove all the clots and leave a cosmetically presentable wound [3, 13].

To conclude, we suggest that understanding the pathophysiology of hemorrhoidal disease guides treatment selection. Appropriate treatment provides symptom resolution in a safe and cost effective manner.

BIBLIOGRAPHY:

1. Milsom JW. Hemorrhoidal disease. In : Beck DE, Wexner SD (eds) Fundamentals of Anorectal Surgery. New York: Mc Grew-Hill 1992: 192-214.
2. Dirckx JH. The biblical plague of "hemorrhoids" Am J Dermatopathol 1985; 7: 341-6.
3. Beck DE. Hemorrhoids. In: Beck DE, Welling DR (eds) patient care in colorectal Surgery. Boston: Little, Brown and company, Inc. 1991: 213-24.
4. Maimonides M. Treatise on Hemorrhoids. Rosner F, Munter S (trans) Philadelphia; JB Lippin Cott 1969.
5. Thompson WHF, The nature of hemorrhoids. Br. J Surg 1975' 62: 542-52.
6. Thulesius O, Gjores JE. Arterio-venous anastamoses in the anal region with reference to the pathogenesis and treatment of hemorrhoides. Acta chir scand 1973; 139: 476-8.
7. Stelzer, F; Staubesand, J, and Machleidt, H: Das corpus cavernosum recti- Die Grundlage der inneren Hemorrhoiden. Legenbecks Arch. Klin. Chir; 299: 302, 1962.
8. Burkitt DP, Graham-Stewart CW. Hemorrhoids-postulated pathogenesis and proposed prevention. Postgrad Med J 1975;51: 631-6.
9. Haas PA, Fox TA, Haas GP. The pathogenesis of hemorrhoids. Dis colon Rectum 1984; 27: 442-50.
10. Johansson JF, Sonneberg A. The prevalence of hemorrhoids and chronic constipation: an epidemiologic study. Gastroenterology 1990; 98: 380-6.
11. Hancock BD. Internal sphincter and the nature of hemorrhoids. Gut 1977; 18: 651-5.
12. Nelson RL, Abcarian H, Davis FG, Perrky V. Prevelance of benign anorectal disease in a randomly selected population. Dis colon Rectum 1995;38: 341-4.
13. Beck DE. Hemorrhoids. In. Beck DE (ed) Handbook of colorectal surgery. St louis: Quality Medical publishing 1997: 299-311.
14. Larach S, Cataldo TE, Beck DE. Non-operative treatment of hemorrhoidal disease. In: Hicks Tc, Beck DE, Opelka FG, Timmcke AE, Complications of colon and Rectal Surgery. Baltimore: williams and wilkins 1997: 173-83.
15. Yang R, Migikovsky B, Peicher J, Laine L. Randomized, prospective trial of direct current versus bipolar electrocoagulation for bleeding internal hemorrhoids. Gastrointest Endosc 1993; 39: 766-9.
16. Lord PH. Diverse methods of managing hemorrhoids dilatation. Dis colon rectum 1973; 16: 180-3.
17. Mazier WP. Hemorrhoids. In: Maxier WP, Levin DH, Luchtefeld MA, Senagore AJ (eds) Surgery of the colon, Rectum and Anus. Philadeplhia: W.B. Saunders Co 1995: 229-54.
18. Allgower M. Conservative management of hemorrhoids III: Partial internal sphincterotomy. Clin Gastroenterol 1975; 4: 608-18.
19. Wilson Mc, Schoefeld P. Cryosurgical hemorrhoidectomy. Br. J Surg 1976; 63: 497-8.
20. Savin S. Hemorrhoidectomy- how I do it. Results of 444 cryorectal surgical operations. Dis colon Rectum 1977; 20: 189-96.

MICROSTOMIA AND "TALAZAWIJ"

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Do not say anything about a person in his absence (which may be true but he would not like to be told in front) — backbite —— ordained to be equal to eating one's dead brother's flesh (Al Quran). "Talazawij" is held in Kashmiri language as a women who is very critic, backbiter and talks excessively to make fault with others. Study of "Microstomia" connotes as a condition with a narrow oral fissure which has no such functional implication as per the available literature. Thus the erroneous belief held about functional aspects of the microstomic mouth related to criticism, backbite and talkativeness seems to have no scientific background. Yet people do the act in this subcontinent although it happens all over the world.

Both the backbiter as well as the listener feel enjoyed over their doings or do so to pass their idle time. The incidence is on the whole far less in the middle east where the listener avoids such hearing because of the teaching fourteen hundred years before and, therefore, always try to change the topic when such sitting occurs.

Speech involves control of respiration by the brain along with control of structures involved in phonation and articulation. Anterior speech cortex (Area 44 and part of 45) is important for speech in general but to understand the meaning of spoken words Association Area of Auditory Cortex (which surrounds the Primary Auditory Cortex (in superior temporal Gyrus and Mescmel's Gyrus) especially area 22 is essential. Besides meaningful listening one needs inhibition of unawanted (here sinful) acoustic stimuli for which Descending Auditory Pathways play the important role. Integrity of such pathways may be gene-dependant which needs investigation and elaboration to explain the governance of nervous control on the concerned structures especially lips of the backbiter. The lips develop as part of the developing face as follows irrespective of sex.

Cells from the primitive streak qastrulate to assemble in the mesenchye ventral to the fore and the hind brain vesicles during the third week of gestation as the prosencephalic and the rhombencephalic organisers. During the fourth week of development these organisers effect the association of five primordia to bound the wide stomodeum (primitive mouth) as prominences known as the frontonasal process (one), two maxillary processes and two mandifular processes. All the five processes contain mesenchyme continous with and containing active centres of growth. These proliferations are actually produced by proliferatron of the neural crest cells and are covered by ectoderm. Majority of the components of bone, cartilage and the ligaments of face and the oral region are contributed from these neural crest cells.

The frontonasal process covers the forebrain ventrolaterally and develop into the forehead, nose, primitive nasal septum, part of the upper lip and primitive palate (Premaxilla). The nerve supplying the frontonasal process and hence, all the derivatives of this process is the first division of the trigeminal nerve i.e. Opthalimic (VI) nerve.

The maxillary processes arise from the dorsocephalic parts of the madibular processes and contribute largely to the formation of cheeks and the upper lip supplied by the maxillary nerve (VII) the nerve which gets incorporated in this process during development as the second division of the trigeiminal nerve.

The mandibular processes are incorporated with the third division of the trigeminal nerve (VIII) and hence all the derivatives from these processes i.e the lowerlip, chin and the lower cheek are supplied by this nerve the mandibular division of the trigeminal (VIII).

A salient groove differentiates the frontal from the nasal parts of the frontonasal process. On the ventrolateral parts of the frontonasal process a thickening appears bilaterally during the fourth week which are the nasal placodes. These placodes, convex in the begining, are the nasal placodes. These placodes, cnvex in the begining are the primordia of the interior nares and the nasal cavity. The mesenchyme surrounding these placodes proliferates as an inverted U-shaped swelling. These placodes sink to form the nasal cavities. The margins of this inverted U (horseshoe) mesenchyme are called the medial and the lateral nasal processes. The maxillary processes grow towards each other and meet the nasal processes. The lateral nasal process is seperated by the nasolacrimal (Nasooptic) groove, in which a solid cord of cells gets burried which, later on canalises to form the nasolacrimal duct. The growing maxillary processes push the nasal processes to raise the

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bridge of the nose. The upper lip and the upper jaw become continuous when the epithelium at the contact points of the maxillary and the nasal processes degenerate. This union seperates the stomodeum (Mouth later on) from the nasal cavity (The vertical height of the philtrum which is very much long "long philtrum" in the Fostal Alcohol Syndrome (FAS) seen in the persons whose mothers consume alcohol during pregnancy. Long philtrum is also seen in foetal dilantin syndrome (children whose mothers consume dilantin for their epilepsy duiring pregnancy) In FAS the upper lip will be thin in addition.

The median nasal process forms the intermaxillary segment and the anterior (ventral) groove of the philtrum of the lip premaxills and its associated gingive and the primary palate.

The maxillary prominence differentiates into the lateral part of the upper lip, maxilla (most of it) and the secondary palate. It is held that the approximating two maxillary processes across and infront of the median nasal process (ventrally) fuse with each other and this is the reaosn that the skin over the philtrum is supplied by the maxillary nerve (The ends of the medial nassal processes are globular — globular processes with a notch between the two which gets obliterated during development to form the single median nasal process — the future bridge of the nose till the tip. It is during this differentation that the mesenchyme from the second arch infiltrates the lips and the cheek to form the muscles of facial expression. Similarly mandibular processes proliferate towards the median line to form the lower lip and the lower cheek. The mesenchyme in the facial prominences also forms various facial bones. The mesenchyme of the first arch, in addition differentiates into the muscles of mastication and some other muscles all of with are supplied by the trigeminal nerve mostly through its mandibular branch (VII).

The lips and the gums form after 6 weeks of intrauterine life. A linear thickening of the ectoderm takes place known as the labiogingival lamine. This lamina grows into the underlying mesenchyme. Later on most of the lamina degenerates leaving the labiogingival (Lip) sulcus which is seen between the lips and the gums. Normally a small area of labiogingival lamina persists in the midline to form the labial frenulum, which attaches the gums to the lips in the midline. The maxillary and the mandibular processes close the angles of the mouth by proliferation of their fusion from near the tragus to the final position / site. These processes do not proceed to fuse and this is the reason that the dog has the angle of mouth extending to near the ears. The various poses, the squars, pursestring like, protruding or invertion thinning the lips during conversation are the acquired poses of sophistication an over due sometimes which may have the psychological background. We know a person, who makes a peculair square mouth while listening to somebody exposing almost twelve teeth in the middle of the rectangular tray, has no apparent developmental error. The ectoders covering the first phyryngeal arch develops into the skin covering the jaws besides the tragus. Along the edges of the lips the ectodermal cells proliferate into the adjacent mesenchyme. These very cells later on disintegrate to form the alveolalabial sulcus which separates the lips from the gums. The inner lining of the sulcus forms mucosa of the gums of both the jaws and also differentiates into the dental lamina from which the teeth develop. Tooth development is a continuous process starting from six weeks of intra uterine life to years after birth.

The pinae develope from six hillocks bordering the dorsal part of the first ectodermal cleft around the external auditory meatus by the end of five weeks of intra uterine life. In first arch syndrome (Mandibulofacial dysostosis) the external ear in deformed along with defects of the lower lids, malar hypoplasia, with down slanting palpebral fissure. This mandibulofacial dysostosis (Treacher Collins Syndrome) is one presentation of the First Arch Syndrome. In the second type known as pierre Robin Syndrome there is hyposlasia of the mandible, claft palate with defects of the ear and eye. Both the conditions are due to insufficient migration of neural crest cells into the first arch during the fourth week. Treacher Collins Syndrome is caused by an autosomal defect. The third manifestation is known as Robin morphological complex where there is a small mandible (Micognathia) and bilateral cleft palate. Congenital microstomia occurs due to over fussion of mardibular with maxcillary process.

It, therefore, connotes that the normal development of the oral fissure depends upon the normal influence of the concerned organisers, normal migration of theneural crest and mesenchymal cells of the second arch, their proper differentiation under normal conditions, Genetic, environmental dietary and drug effects need to be searched for. However small oral aperture or thinner lipped talkatives can not be explained on the basis of embryology in both the male as well as female.

Further Readings:

1. Behraman R.E. (ed): Nelson Textbook of Paediatrics, 14th ed. Philadelphia W.B. Saunders, 1992.
2. Bijlani R.L. Understanding Medical Physiology Ist Ed. Jaype Publishers 1995;pp.861-62.
3. Berkovitz BKB and Moxham B.O. A text book of Head and Neck Anatomy Ed. Worfe Medical Publication Ltd. 1998.
4. Gerlin R.J. Cohen Jr. M.M. Lewin S.S. Syndrones ofthe Head and Neck, 3rd ed. New York, Oxford Uni Press, 1990.
5. Guyton BC and Hall J.E. Text book of Medical Physiology Ed. 10 W..B. Saunders 2000.
6. Grays Anatomy. The Anatomical bain of Medicine and Surgery Ed. 38 Churchill Livingstone 2000.
7. Kirby M.F. Bockman DE: Neural Crest and Normal Development.

a new perspective. Anat Rec 1984;209:1.

8. Latham RA: Development and structure of the Premaxillary Deformity in Bilateral Cleft Lip and Palate. Br. J. Plas Surg. 1973;26:1-11.
9. Latham RA, Deaton TO: The structural Basis of the Philtrum and the Contour of the Vermilltion Border: a study of the musculature of the Upper Lip. J. Anat 1976;121:151-160.
10. Moore KL: Clinically Criented Anatomy, 3rd ed. baltimore, William and Wilkine, 1992.
11. Orbans Oral Histology and Embrylogy Ed. 10. CBS Publishers and distributors.
12. Rinrichesen K: The Early Development of Morphology and patterns of the Face in the Human Embryo. Advances in Anatomy, Embryology and cell Biology 98. New York, Springer Verlag, 1985.
13. Sandham A, Nelson r, Embryology of the Middle third of the Face. Early Human Development 1985;10:313.
14. Wedden SE, Raphe JR, Tickle C: Pattern Formation in the Facial Primordia, Development 1988;103 (suppl):31.

UTILITY OF FINE NEEDLE ASPIRATION CYTOLOGY IN THE DIAGNOSIS OF PRIMARY MALIGNANT BONE TUMORS (PART I)

Khan AR, MD.

Abstract: *To review of efficacy of FNAC in the diagnosis of primary malignant bone tumors, a retrospective analysis over a period of 11½ years (January, 1991 -June 2002) revealed 110 bone tumors including 54 primary bone malignancies. Histopathology was available for comparison in all the cases except in the 12 cases of Myeloma aspirated from the Sternum. Out of primary bone malignancies Myeloma was the most common (20 cases) followed by Osteo-sarcoma (12 cases), Ewing's sarcoma (10 cases), NHL (6 cases), Adamantinoma (1 case) and Chordoma (1 case). There was 100% Cyto-histopathologic co-relation in all but one case of Osteo-sarcoma (false negative). Thus it was concluded that FNAC of osteolytic primary malginant bone tumours in the context of clinical and radiology background in the experience hands by a pathologist familiar with bone pathology is very useful in the pre-operative diagnosis*

Key Words: *FNAC, Bone tumours Primary malignant bone tumours*

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Introduction

Fine needle aspiration is now widely accepted diagnostic modality. With advancement in imaging techniques virtually any lesion can be targeted. Though bone FNAC was attempted in early 1930's[1,2] yet somehow it could not keep pace with FNAC at other sites. With the management of some primary bone malignancy by pre-operative chemotherapy and its advantages over open needle bone biopsy, FNAC is bound to gain popularity.

In this article author discusses experience of FNAC of primary bone malignancy over the last 11½ years (Jan 1991-June 2002).

Material and Methods

A retrspective analysis over the last 11½ years (Jan 1991-June 2002) revealed 110 bone tumors. More than 90% of the cases were from the clinic of the author and the remaining 10% were reported by the author in the Pathology Department Govt. Medical College, Srinagar. The aspirations were conducted with the help of radiographs and in a few cases help of Orthopedic surgeon and radiologist was sought. A 20 ml disposable syringe with and without syringe holder with 23-18 guage needles were used at non-sternal sites. The sternal sites were aspirated with conventionsl bone marrow aspiration needle. The material aspirated was smeared on to 5-8 slides MGG was used regularly on 2 slides, haematoxyin and eosin on many slides P.A.S in selected cases and 1-2 slides reserved for any special studies.

Results

The anlysis revealed 110 bone tumors (table-1)

Myeloma (MM)

As shown in the table-2, 12 of the 20 cases were aspirated from the sternum. They had suspicion of myeloma clinically. Remaining 8 cases were aspirated from non-sternal sites including ribs (2), clavicle (2), upper ends tibia (2), maxilla (1) and ileum (1). These 8 cases at the time of presentation had localised pain and lytic lesions. The age of patient ranged between 45 and 67 years with 17 males and 3 females.

Cytological findings

Smears showed pedominantly or exclusively of plasma cells both mature and immature and crowding out of other marrow elements. Tumor cells were well spread in both sternal and FNAC aspirates. However cell clusters were not uncommon in addition in FNAC aspirates at non-sternal sites. The mature cells showed abundant basophilic cytoplasm round eccentric nucleus with coarse clumpy cartwheel pattern chromatin and paranuclear hof. Some cells contained 2 to 3 nuclei. The immature plasma cells showed a high nucleo-cytoplasmic ratio, granular non-cartwheel type chromation and 1 - 2 prominent nucleoli. (Figs.1-3). The diagnosis of myeloma was confirmed in eight cases by incisional biopsy and in remaining 12 cases aspirated from sternal sites by other investigations.

Osteo-sarcoma (O.S)

There were 12 cases of Osteosarcoma with age range of 10-35 years, including 8 males and 4 females. The anatomical sites included 8 around knee (5 from tibia. 1 from fibula, 2 from femur). 3 from humerus (2 from lower

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end and 1 from upper end) and 1 from scapula (Table-3).

Table 1: FNAC of 110 Primary Bone Tumors (1992 – 2002)

(I)	**Primary**	
A.	**Benign**	
	1. Chondroma	10
	2. Osteo- Chondroma	04
	3. Giant cell Tumor	13
	Total	**27**
b.	**Malignant**	
(a)	**Primary.**	
	1. Myeloma	20
	2. Osteo-sarcoma	12
	3. Chondro- Sarcoma	04
	4. E-S/PNET	10
	5. Non-Hodgkin's *Lymphoma*	06
	6.. Chordoma	01
	7. Adamantinoma	01
	Total	**54**
(II)	Metastatic	29
	Grand Total	110

Table 2: FANC-Myeloma (M M) Bone, Non- Sternal Sites

S.No	Age / Sex	Anat Site	FNAC	Histopath
1.	65/M	Ribs	Myeloma	Myeloma
2.	50/M	Ribs	Myeloma	Myeloma
3.	50/F	Clavicle	Myeloma	Myeloma
4.	55/M	Tibia-UE	Myeloma	Myeloma
5.	67/M	Tibia-UE	Myeloma	Myeloma
6.	60/M	Maxilla	Myeloma	Myeloma
7.	57/M	Ileum	Myeloma	Myeloma
8.	54/M	Clavicle	Myeloma	Myeloma

Sternal Site (12 cases)

		(Age in years)
M	(10)	45,60,51,54,46,54,67,61,64,65
F	(2)	52, 47

Sex M = 17
F = 03
Total 20

Abbr. MM Multiple Myeloma, UE Upper End

Cytological findings

11 cases had adequate material in the smears and no material was seen in one case. Smear pattern in these 11 cases was that of the pleomorphic sarcoma. Tumor cells were seen dispersed and in small and large fragments amounting to microbiopsies. Spindle, polyhedral and small cells in variable mixture with abundant to scanty dense cytoplasma showed round to spindle to polypoid hyperchromatic nuclei with irregular dense nuclear membrane and 1 or more prominent nucleoli. Varying numbers of both tumor and osteoclast type giant cells were present in all the cases. Tumor cells were larger in size than mononuclear counterpart, with 1-3 lobulated hyperchromatic nuclei and abundant dense cytoplasm. Mitotic figures were occasionally seen. Stroma consistent with osteoid was present in all the cases from scanty to abundant as magenta to a redish homogenous and fibrillar material either dispersed or in fragments intermingled with tumor cells (Fig.4-5). With the clinical and radiological picture, all the 11 cases were confidently diagnosed as osteosarcoma. Histopathology was avialable in all the cases and confirmed the diagnosis of conventional osteosarcoma.

Table 3: FNAC- Osteo- Sarcoma (O. S) Bone (12 Cases)

S.No.	Age/ Sex	Anat Site	FNAC	Histopath
1.	20/ M	Fibula-U.E	O.S	O.S
2.	35/F	Tibia- U.E	O. S	O .S
3.	16/M	Tibia- U.E	O. S	O.S
4.	16/F	Humerus-U.E	O. S	O.S
5.	15/M	Tibia- U.E	O. S	O .S
6.	16/F	Femer- U.E	O. S	O. S
7.	25/F	Tibia-U.E	Dry-Smears	O. S
8.	16/M	Tibia -U.E	O. S	O. S
9.	20/F	Humerus-U.E	O. S	O. S
10.	10/F	Scapula	O. S	O. S
11.	20/F	Femur-U.E	O. S	O. S
12.	20/F	Humerus-U.E	O. S	O. S
Sex	M/ 4 F/ 8	Age 10 - 35		

Abbr.O.S. :- Osteosarcoma, U.E :- Upper End, L.E :- Lower End

Chondro-sarcoma (C.S)

There were 4 cases of chondrosarcoma. These included 40 year male, 57 year male, 25 year male and 42 year female with tumors arising from the upper end of humerus, metacarpals, iliac bone and neck of femur respectively (Table 4).

Table 4: FNAC- Chondro - Sarcoma (C.S) Bone (4 Cases)

S.No	Age/Sex	Anat Site	FNAC	Histopath
1.	40/M	Humerus-U.E	C.S –agg	C.S grade 2
2.	57/M	Metacarpal	C.S-agg	C.S grade 2
3.	25/ M	Ileum	C.S –agg	C.S grade 2
4.	42/F	Femur-U.E	C.S low grade	C.S grade 1

Abbr :- C.S. Chondrosarcoma, AGG. Aggressive, U.E. Upper end

Cytological findings

Abundant materials was present in the smears in all the 4 cases. In the first three cases, the smear pattern was almost identical, with abundant purplish homogenous and fibrillar granular material dispersed and in fragments. The tumor cells widely scattered within this stoma were round with little variation in size and shape with round nuclei, small nucleoli and moderate cytoplasm. Some bi-nucleated cells were also present. A clear halo was seen around some cells. No mitatic figures or necrosis was seen in any of the cases. With the clinical data and radiographs, smears were reported as consistent with aggressive chondro-sarcoma (Figs.6-7) in all the 3 cases.

Table 5: FNAC- Ewing's Sarcoma (E.S) / PNET Bone (10 Cases)

S.No.	Age/Sex	Anat Site	FNAC	Histopath
1.	10/M	Scapula	E.S.	E.S.
2.	18/M	Ileum	E.S.	E.S.
3.	20/M	Ileum	E.S.	E.S.
4.	15/M	Ileum	E.S.	E.S.
5.	17/F	Ribs	ES-PNET	PNET
6.	16/M	Ribs	E.S.	E.S.
7.	22/M	Ileum	E.S.	E.S.
8.	11/M	Tibia	E.S.	E.S.
9.	4 ½	Scalp	E.S.	E.S.
10.	15/M	Ribs	E.S.	E.S.
Sex	M – 8	Age 41 ½ - 22		
	F - 2			

In case 4, a 40 female with a soft tissue mass in right inguinal region and destruction of the neck of the femur, smears showed abundant mucinous material and granular chondroid matrix and very few benign chondrocytes suggesting the diagnosis of chondroma (Fig.8). However, with radiographs, the suggestion of lower grade chondro-sarcoma was made. Histopathology revealed chondro-sarcoma — grade 1.

Table 6: FNAC – Non-Hodgkins Lymphoma (NHL) (6 Cases)

S.No	Age/Sex	Anat Site	FNAC	Histopath
1.	30/F	Femur – L.E	?NHL	LCL
2.	70/M	Radius –U.E	LCL	LCL
3.	40/F	Ribs +soft tissue	LCL	LCL
4.	65/M	Ischium	LCL	LCL
5.	45/M	Ribs–Soft tissue	Mixed all	LCL
6.	70/M	Ileum	LCL	LCL
Sex	M 4	Localised	1,2	(Stage 1)
	F 2	Disseminated	3-6	(Stage 4)

Ewing's Sarcoma (E.S)- PNET

As seen in the Table 5, there were 10 cases of E.S. with age range of 4 1/2 - 22 years, including 8 males and 2 females. Their primary anatomical sites were ileum (4) ribs (3), scapula (1), scalp (1) and tibia (1). There were 2 cases of metachronous lesions, in ileum and abdominal wall (case no. 7), and tibia and scalp (case no. 8). In addition there was a case of local recurrence in ribs (case no. 6). They presented as local pain and lytic lesions (9 cases) and chronic osteomyleitis tibia (1 case). Constitutional symptoms were present in three cases.

Cytological Findings

Smears (Figs.9-12) yielded adequate material at all sites primary, metachronous and recurrent. Smears were highly cellular with striking monotony of cells devoid of stroma, mostly dispersed, with some loose clusters, and with occasional nuclear molding. There was one case with many rosettes labeled as PNET (case no. 5). Two types of tumor cells were seen. The larger pale cells were predominant with fine granular chromatin, scanty pale to vacuolated cytoplasm with occasion cytoplasmic blistering. These cells showed variable PAS positivety. The small darker cells, were infrequent, with smaller darker nuclei and scanty cytoplasm. Mitotic figures were infrequent (Fig.9-12). Histopathology was available in all cases which confirmed the cytology diagnosis.

Non-Hodgkin's Lymphoma (NHL)

There were 6 cases of NHL, with age range of 30-70 years, with only one case below the age 40 (Table-6). There were 4 males and 2 females. The anatomical sites were, ribs 2 ileum 1, ischium 1, lower end of the femur 1 and upper end of radius 1. Soft tissue mass was present in 2 cases with rib involvement. X-ray showed lytic lesions in all. When investigated subsequent to FNAC diagnosis only 2 lesion (femur and radius) showed localised disease. The other 4 proved to be cases of disseminated lymphoma who primarily presented with bone involvement. Histopathology of excised lesions was availble in all the cases.

Table 7:FNAC- Chordoma + Adamantinoma (2 Cases)

S.No	Age/Sex	Anat Site	FNAC	Histopath
1.	40/M	Sacrum	Chordoma	Chordoma
2.	40/M	Tibia	Adamantinoma	Adamantinoma

Cytologic Findings

Adequate material was present in all the cases. The cytological criteria for diagnosis were same as for lymph nodes[3,4]. Thus 4 cases were labeled as large cell and 2 cases as mixed cell lymphomas. The lymphoid cells were dispersed with large non-cleaved and cleaved cells with varying percentage of small cleaved cells and lymphocytes. Lymphoglandular bodies were present in all the cases (Fig.13). Histopathology revealed large cell non-Hodgkin's lymphoma in all the cases.

Chordoma

One case of chordoma sacrum was recorded in a 40 year old male who presented with local pain (Table 7). X-ray showed destruction of bone with soft tissue extension.

Cytologic Findings

As seen in the (Fig.14-15) abundant material was present in the smears dominated by magenta coloured bubbly fibrillar stroma in MG stain. The tumor cells, were scattered in the stroma as round, uniform cells with uniformnuclei, and pale vacuolated cytoplasm. Some cells contained 2 to 3 nuclei. Occasional cells were larger in size with abundant bubbly cytoplasm with multiple nuclei,

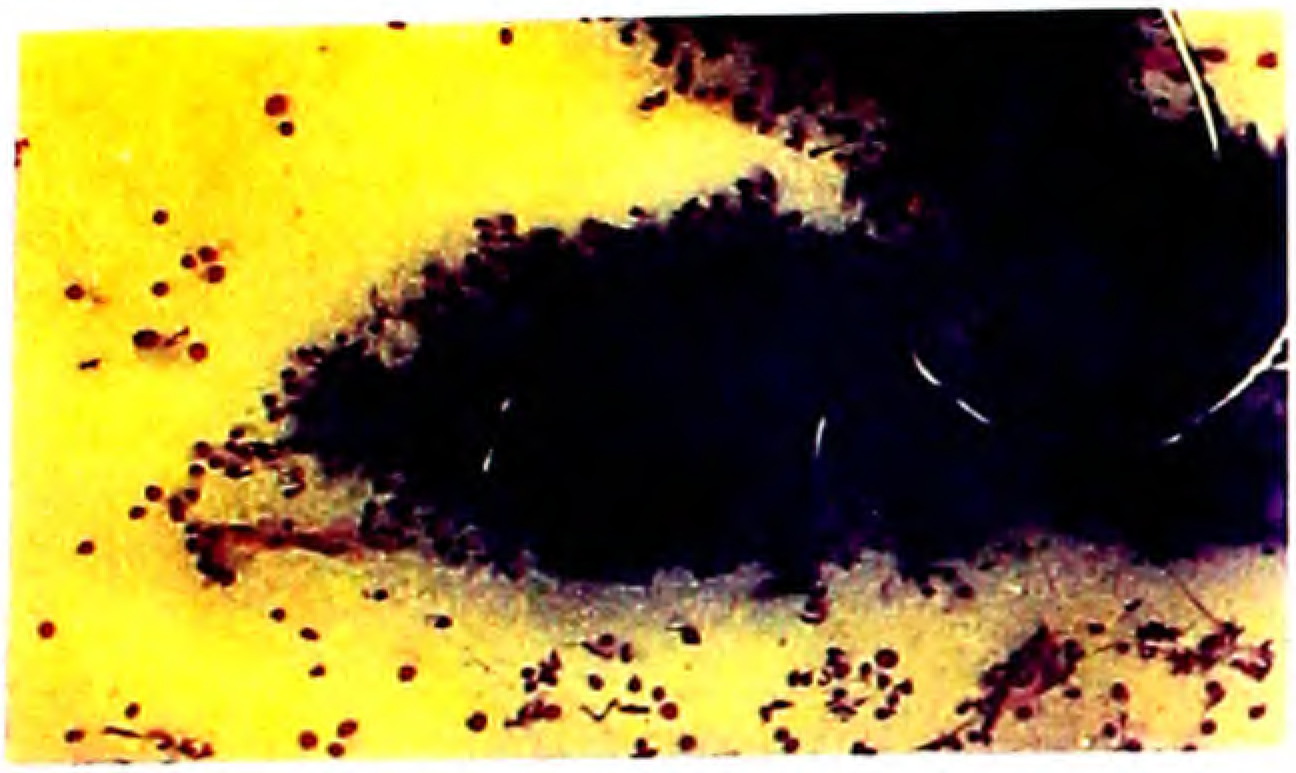

Fig. 1. Myeloma — FNAC tibia, shows a large cluster (micro-biopsy) of tumor cells dispersed plasma cells.

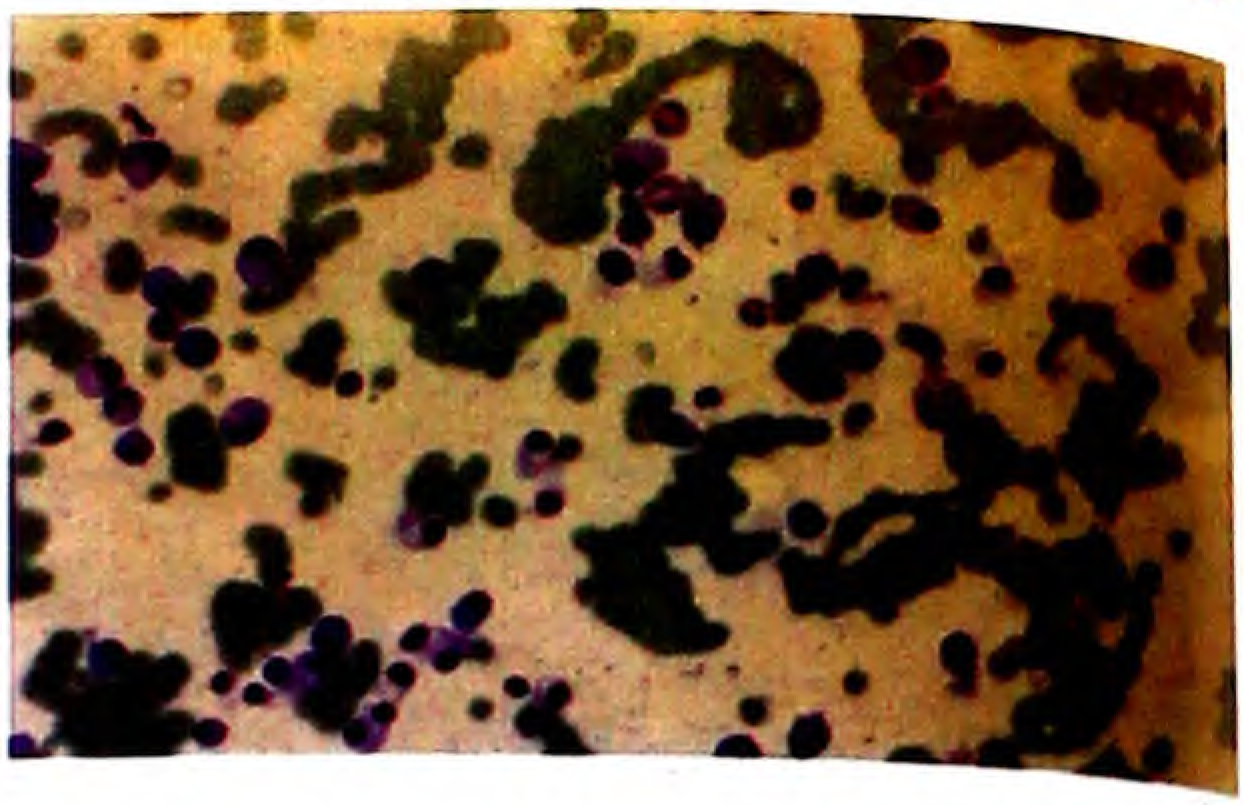

Fig. 2. Another field from Fig. 1, shows dispersed plasma cells. MGG X 100

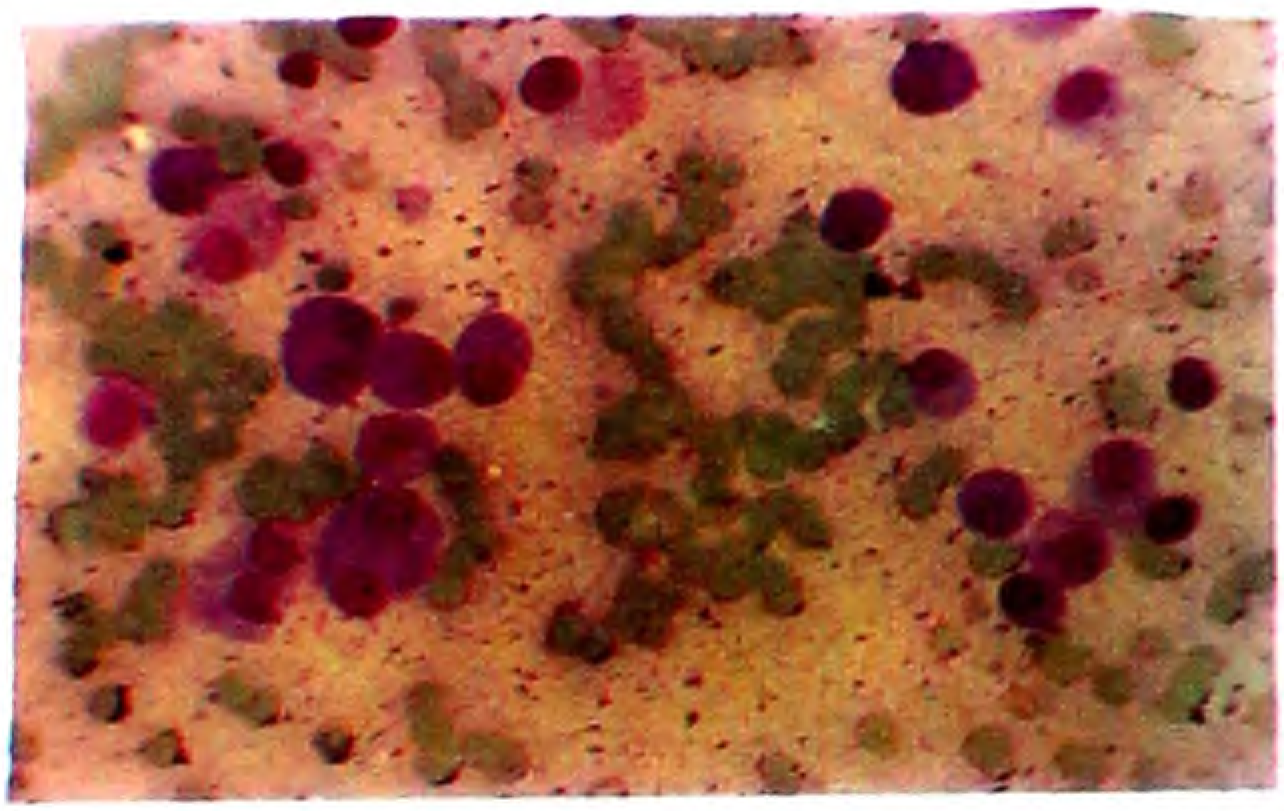

Fig. 3. Myeloma — from sternal puncture shows mature and immature plasma cells. MGG x 400

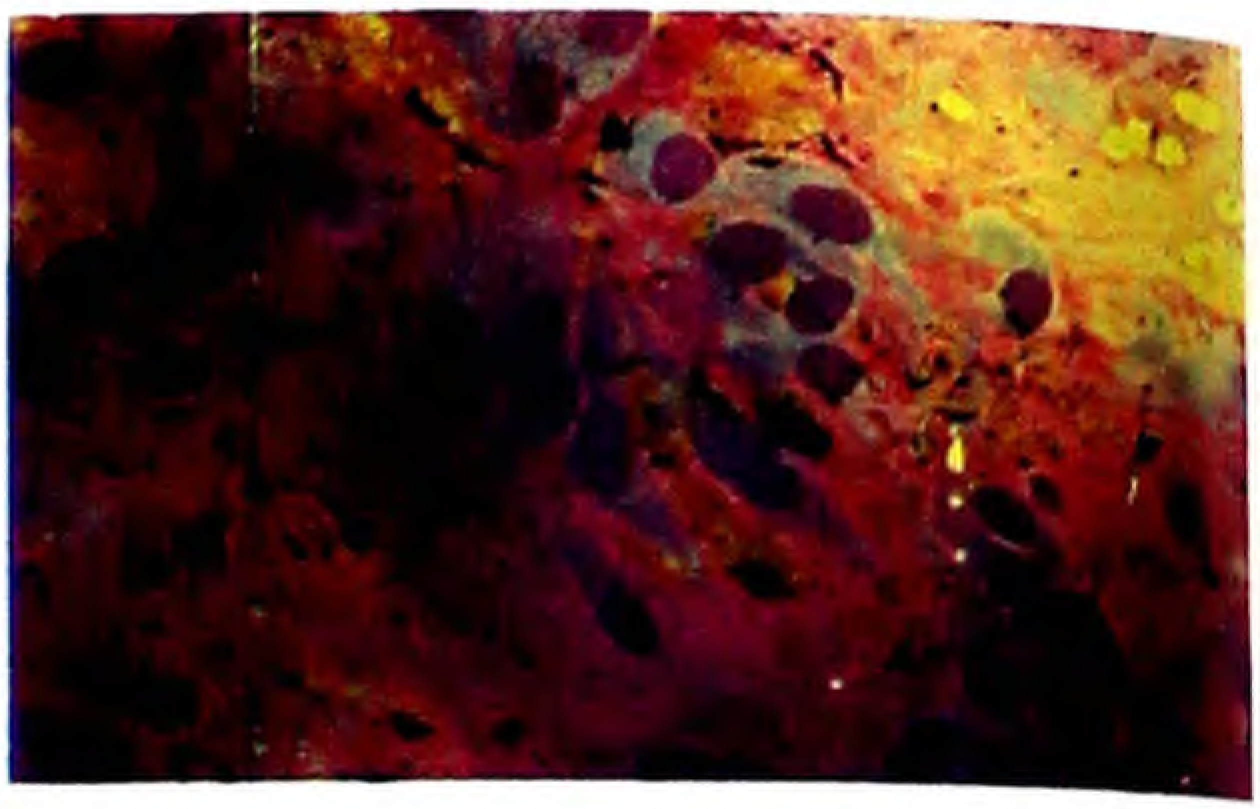

Fig. 4. FNAC — Osteo-sarcoma, shows poly-hedral malignnant tumor cells in abundant pink collagenous matrix interpreted as osteoid. MGG x 400.

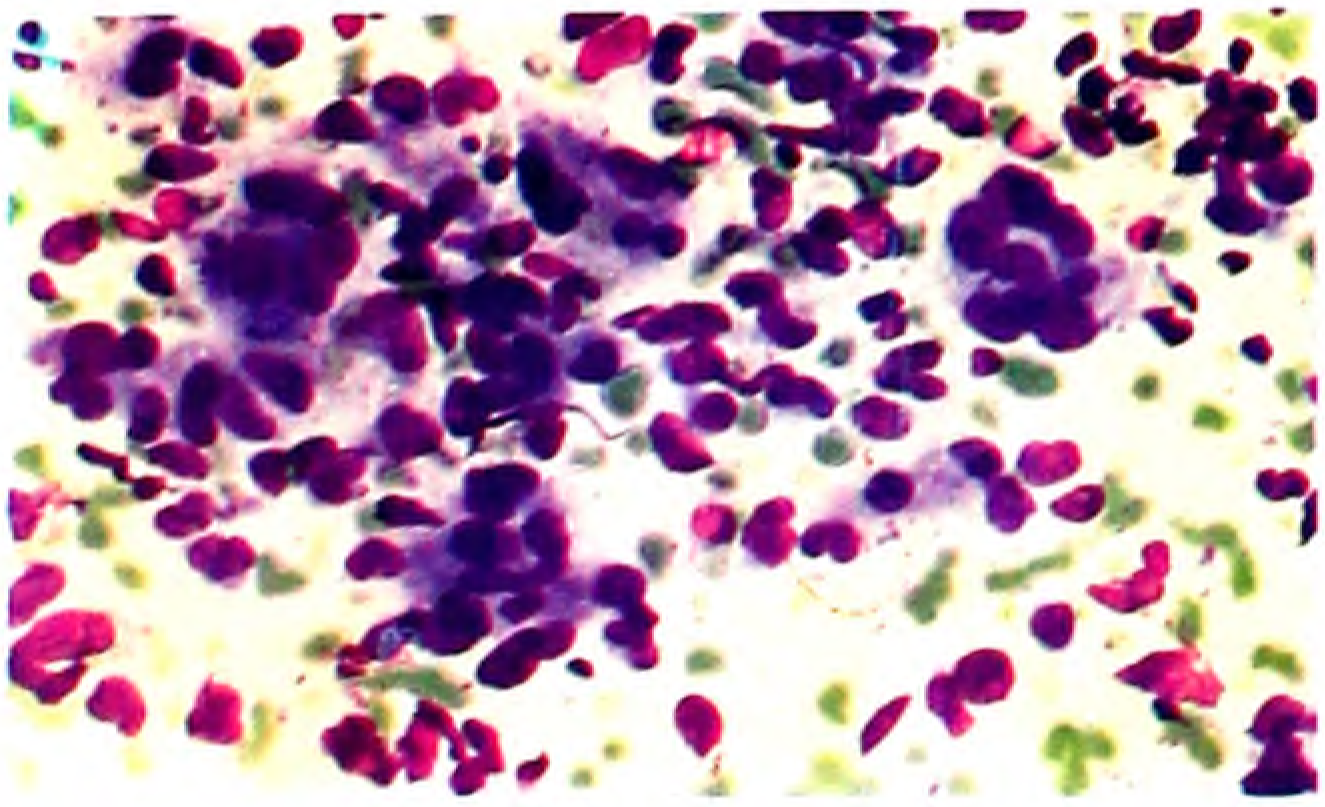

Fig. 5 FNAC — Osteo-sarcoma, with striking pleomorphism of tumor cells and tumor giant cells. No osteroid seen. This pleomorphic tumor without osteoid in context of appropriate age and radiographs is diagnostic of osteo-sarcom. MGG x 400.

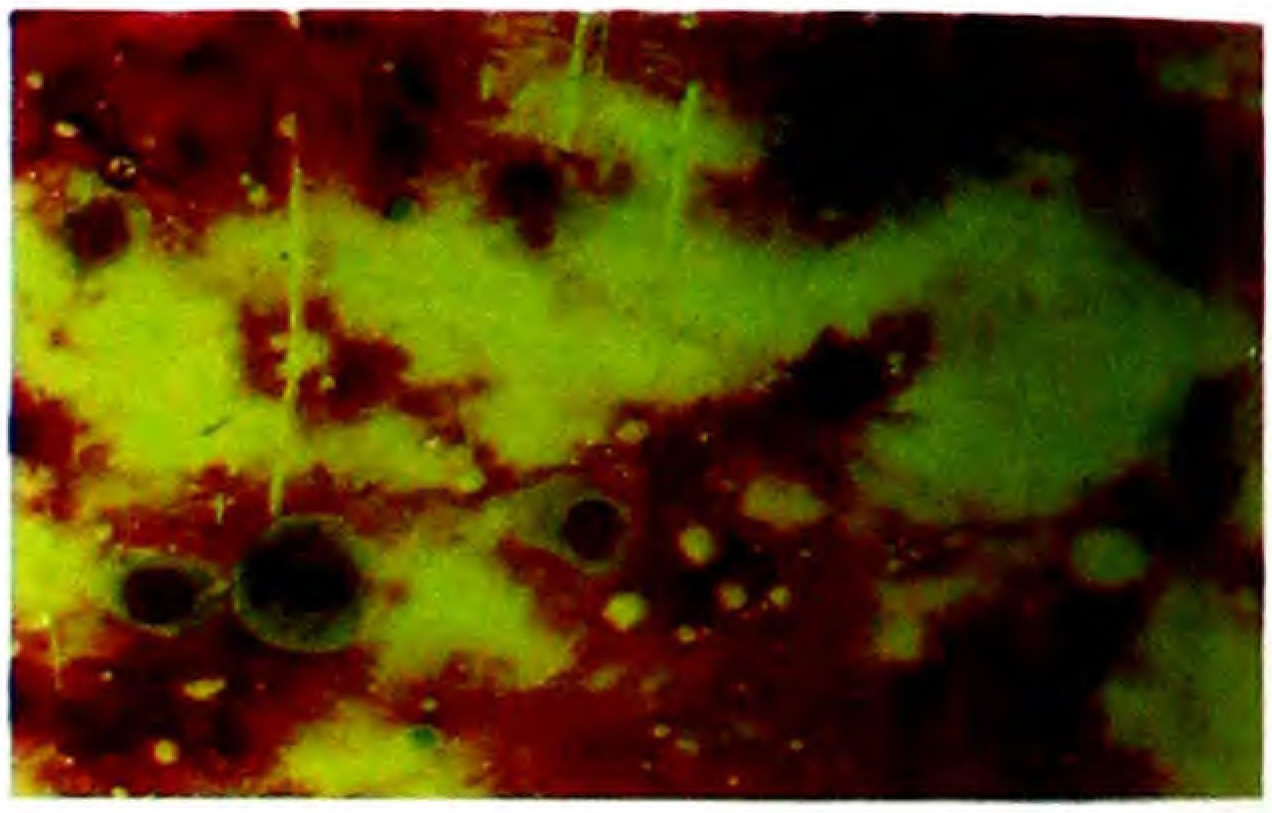

Fig. 6. FNAC — Chondro-sarcoma - aggressive -(Grade. 2) shows large round mono and binucleated cartiage tumor cells with high N/C ratio, embedded in chondroid matrix. MGG x 400.

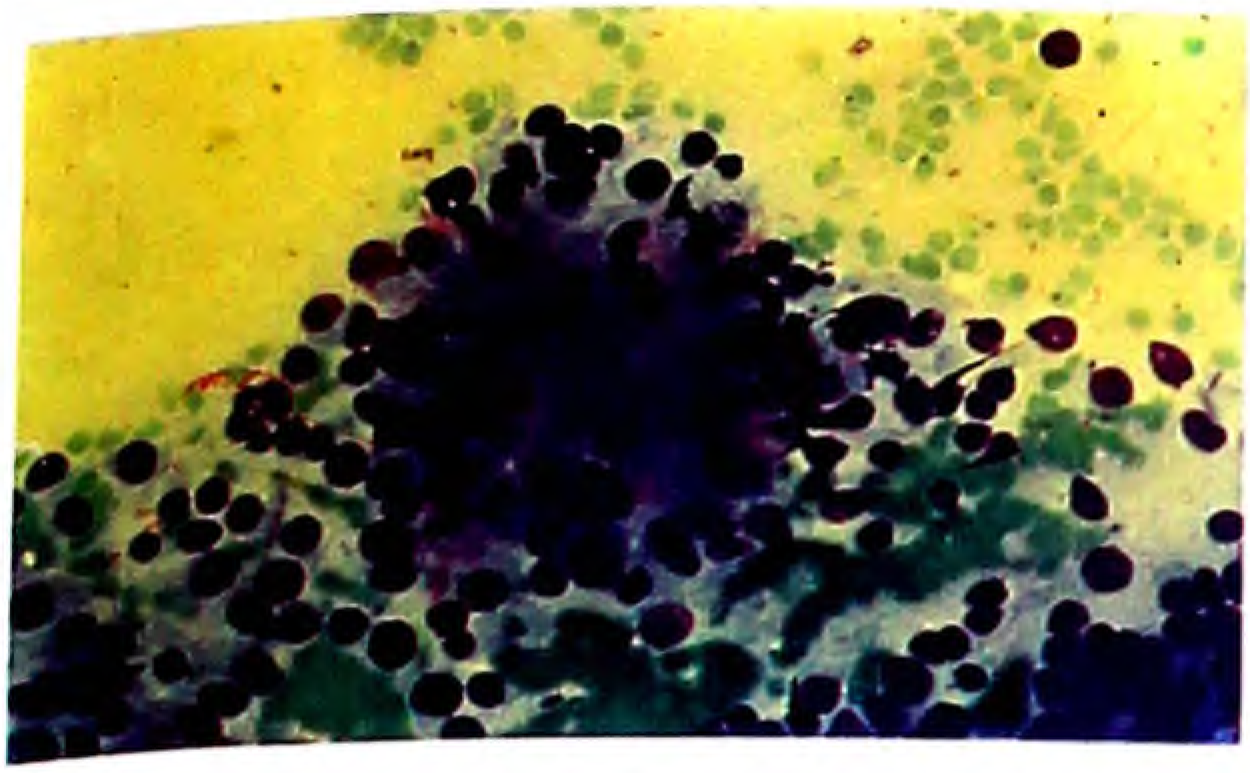

Fig. 7. FNAC — Chandro-sarcoma, aggressive -(Grade 2) shows large round cartilage tumor cells with high N/C ratio dispersed and embedded in abundant magenta coloured chondroid matrix MGG x 400.

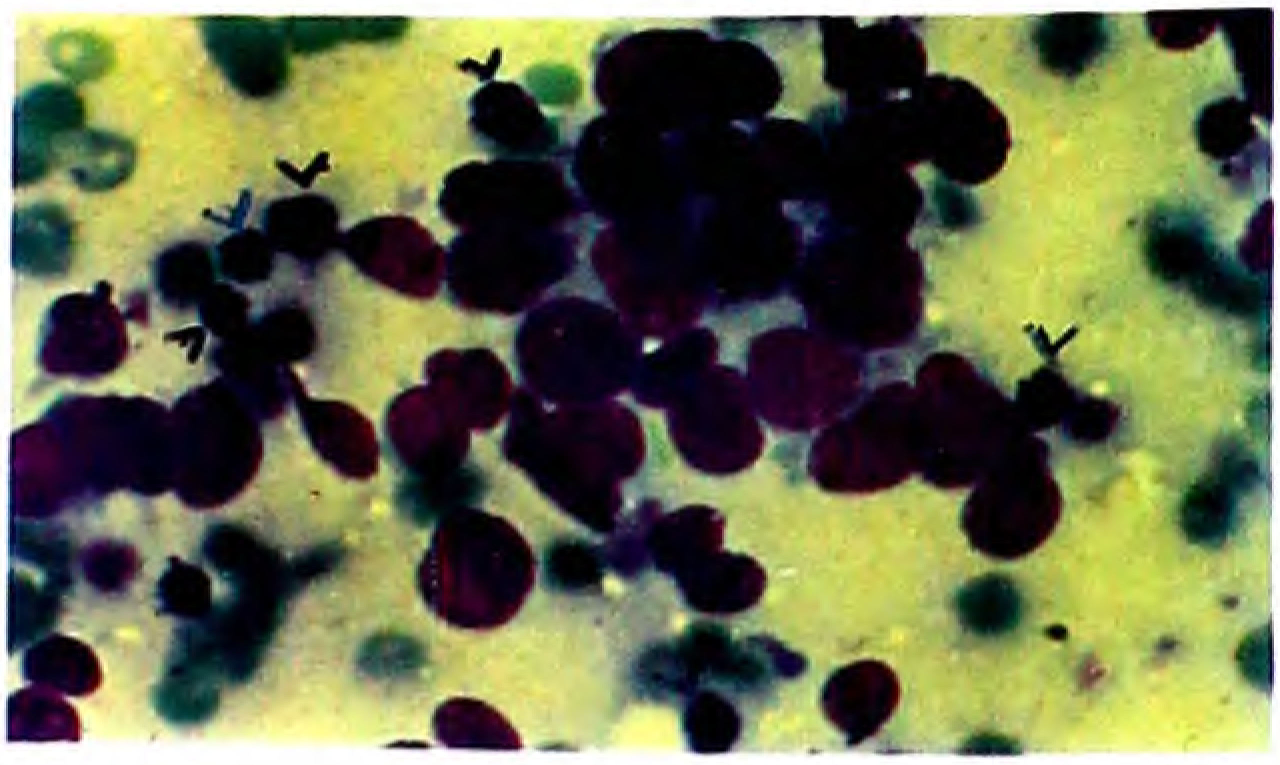

Fig. 8. FNAC — Chondro sarcoma low-grade -(Grade. I) from neck of femer, shows abundant chondroid matrix with sparsely distributed benign looking chondroytes. Only with the help of radiographs interpreted a low-grade C.S. MGG x 200.

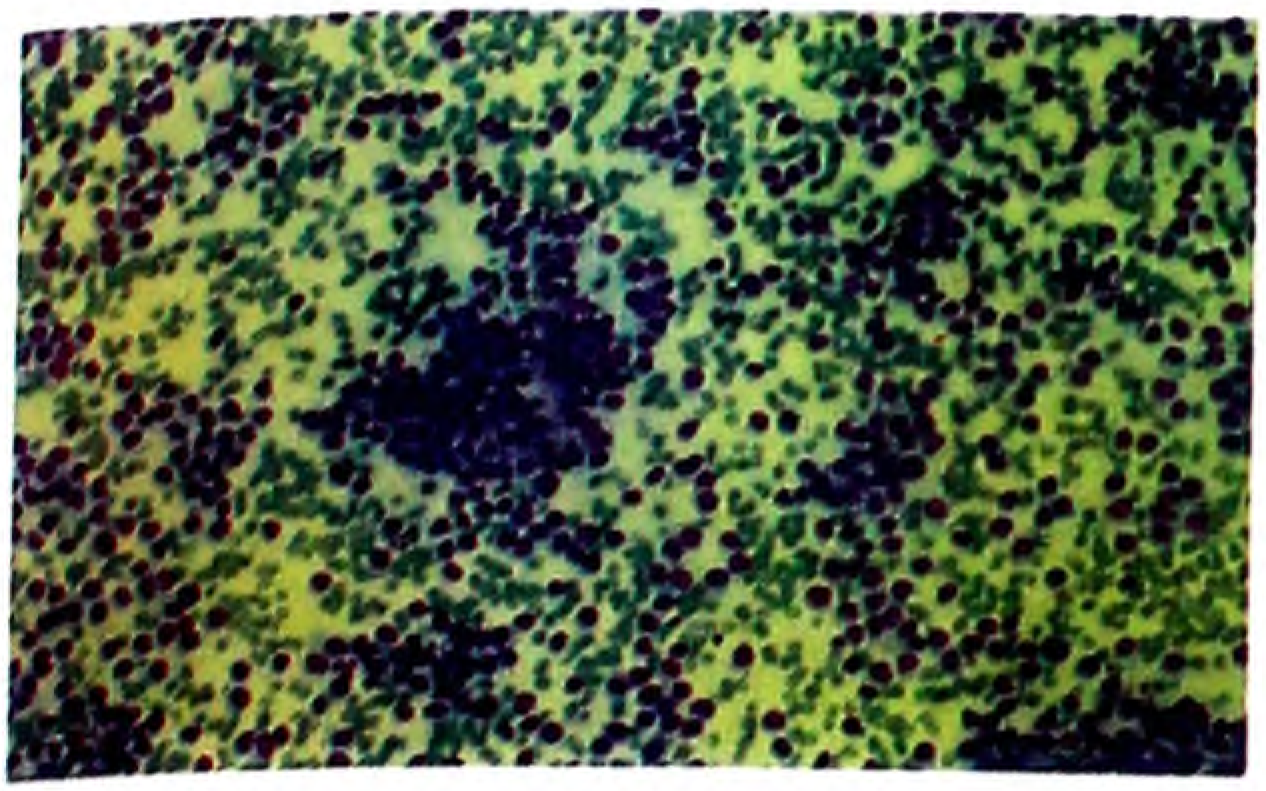

Fig. 9. FNAC — ES-PNET, above shows monomorphic round tumor cells dispersed and in loose clusters, and fine vacuolations of cytoplam MGG x 100

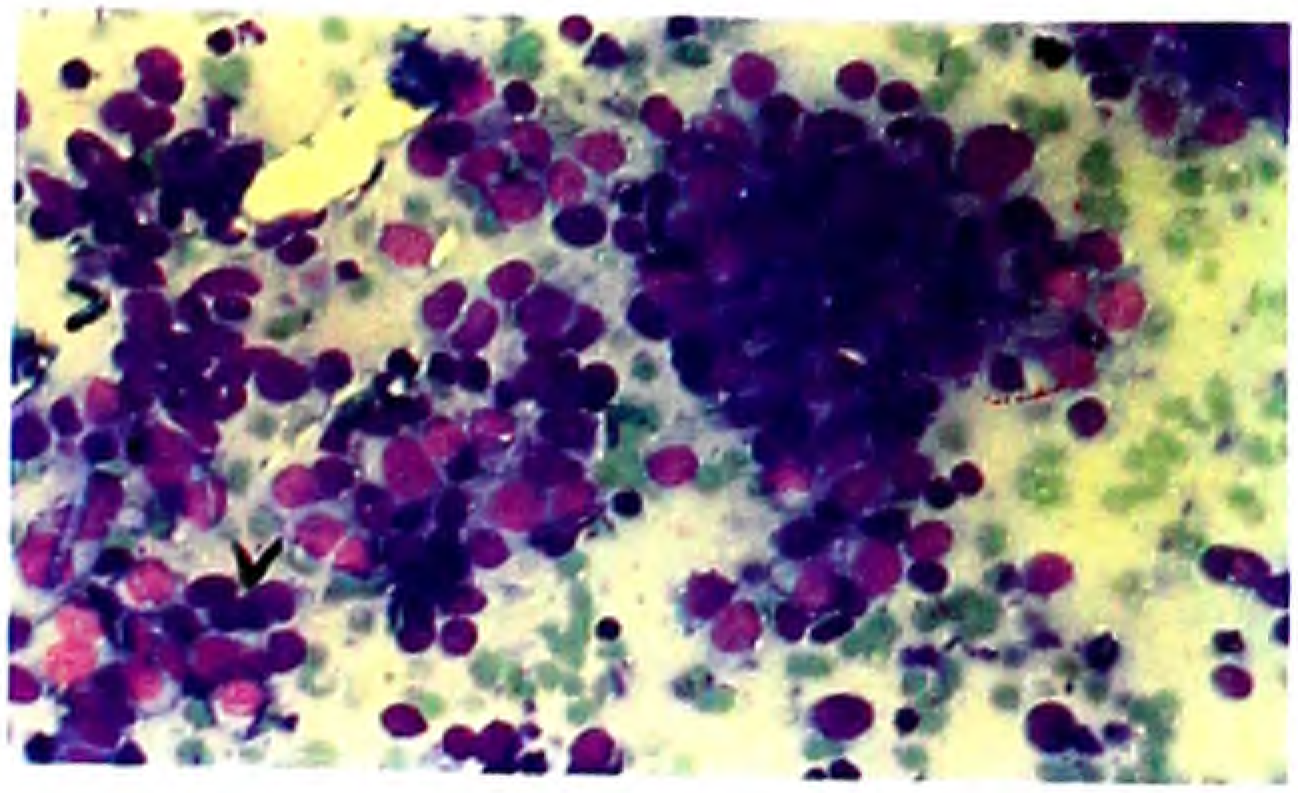

Fig. 10. FNAC — ES-PNET, higher power to show clusters of tumor cells composed of large pale cells and smaller dark cells and nuclear molding - (arrow) MGG x 200

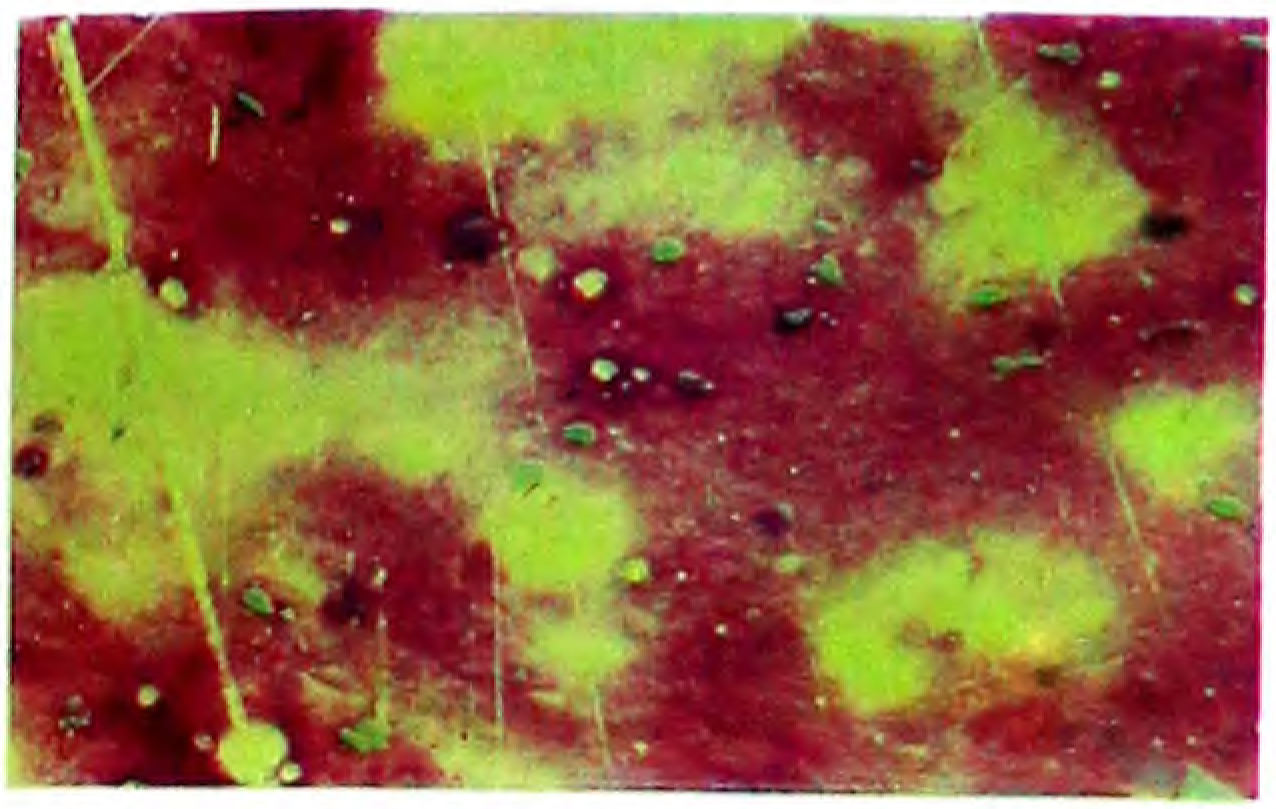

Fig. 11. FNAC — ES-PNET, still higher power to show large pale and small dark cells (arrow) MGG x 400

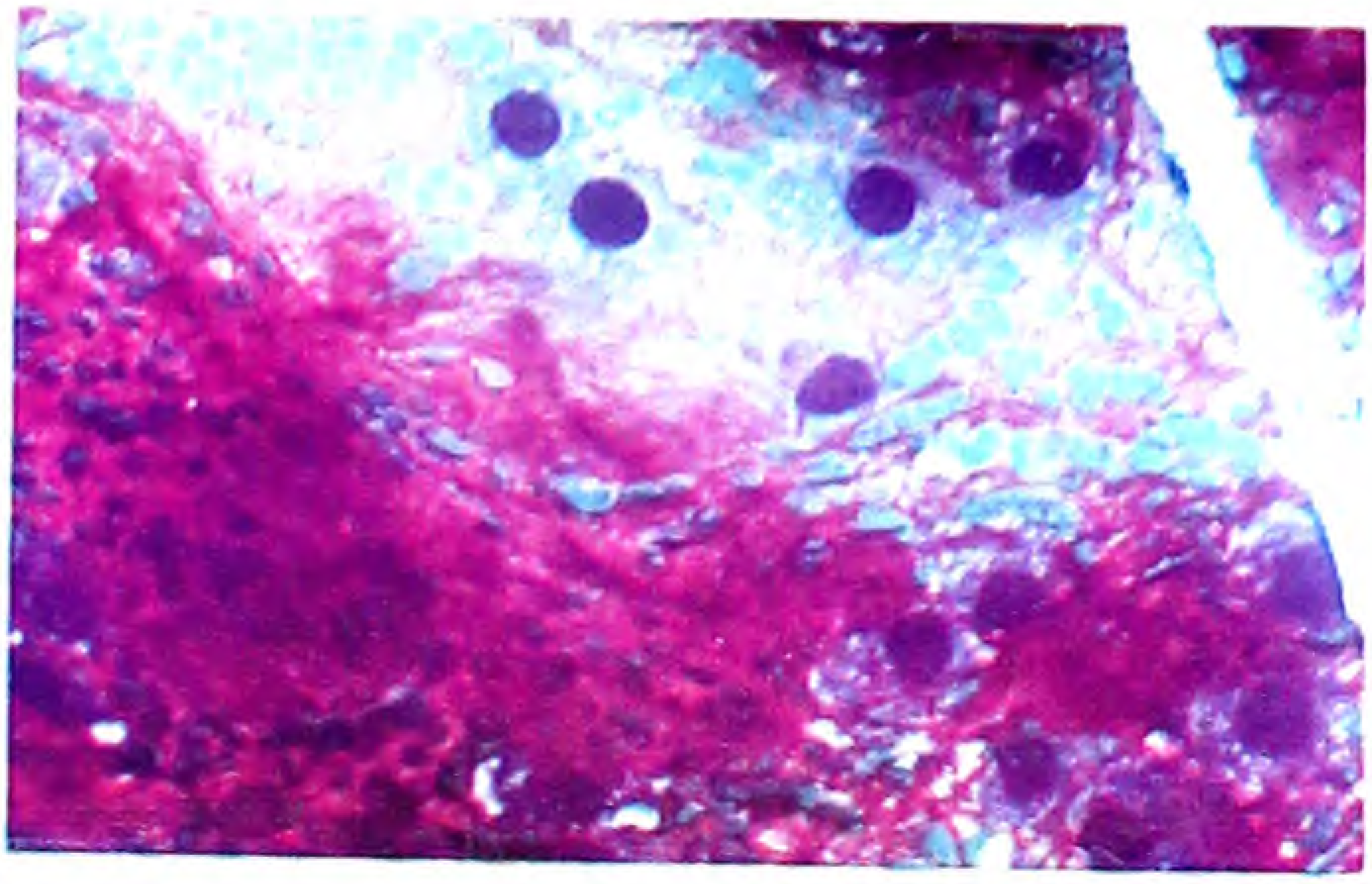

Fig. 12. FNAC — ES-PNET, to show a rosette, with tumor cells aligning around central fibrillar material. MGG x 200

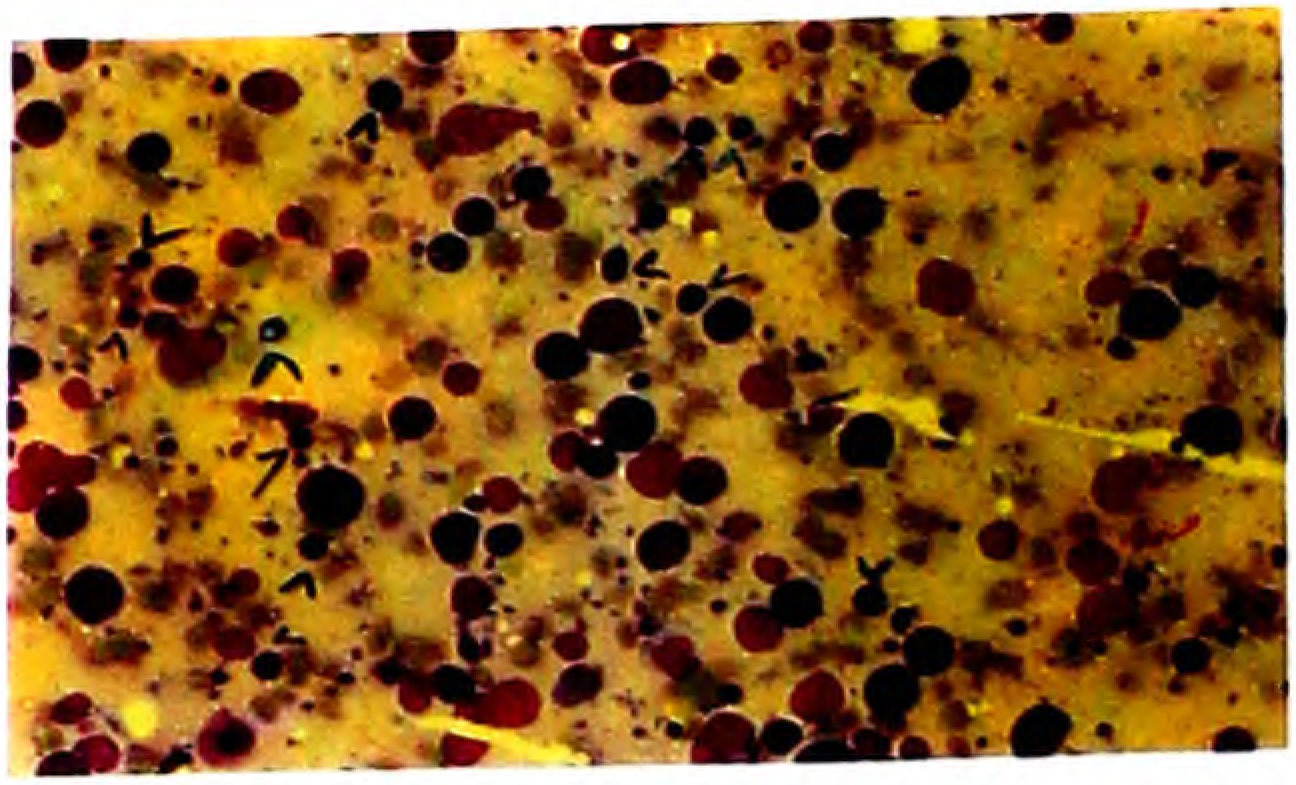

Fig. 13. FNAC, NHL — Iliac bone shows dispersed small and medium sized lymphoma cells, with numerous lymphoglandular bodies - arrows MGG x 400

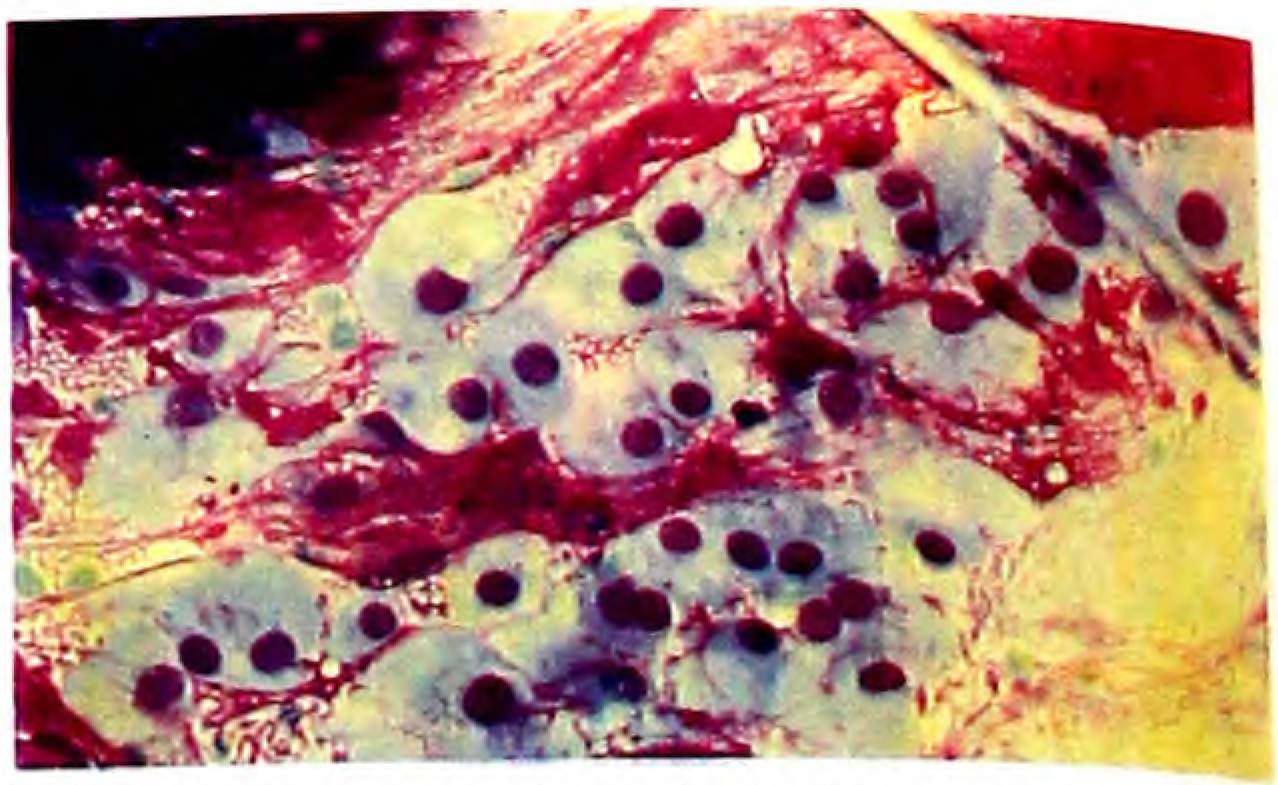

Fig. 14. FNAC, Chordoma-sacrum shows monotonous tumor cells loosely packed due to abundant clear to vacualated cytoplasm and a typically fibrillar stroma, in the background which is otherwise clean MGG x 400

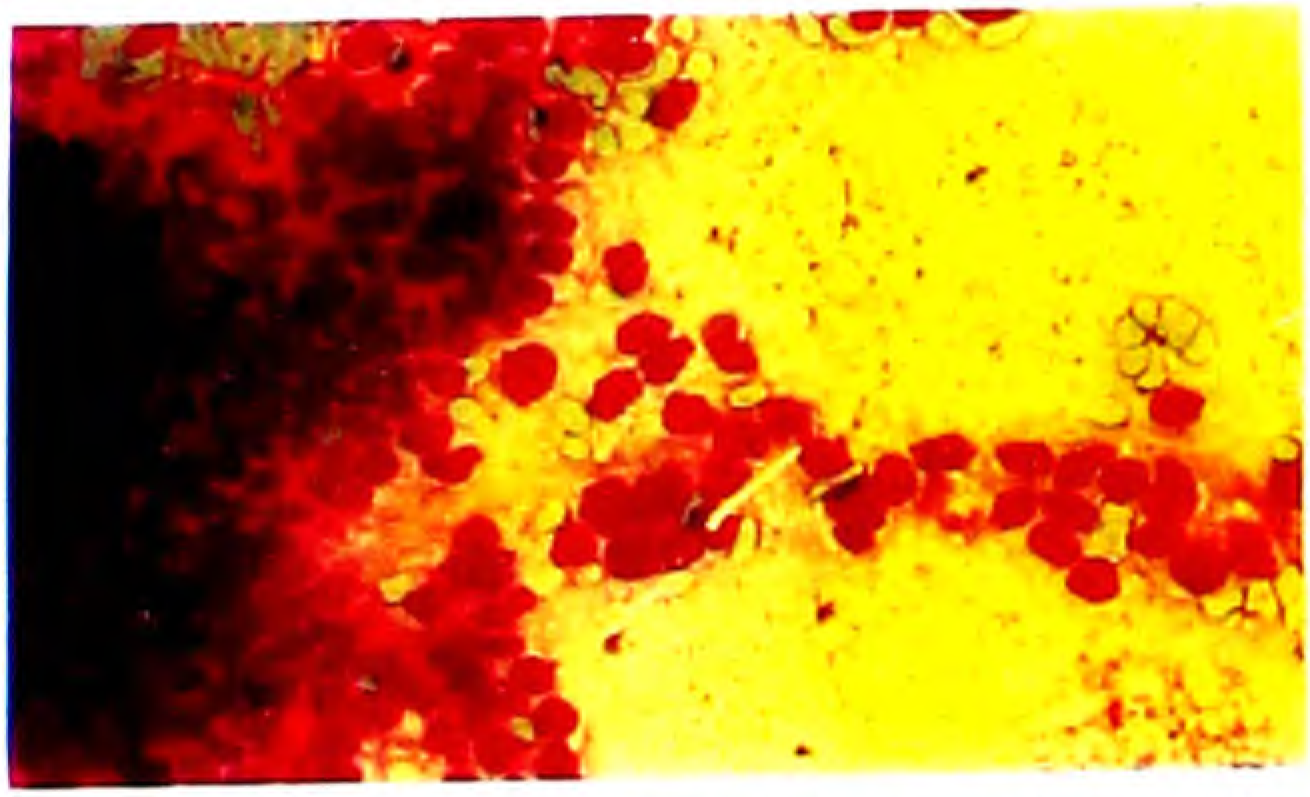

Fig. 15. FNAC, Chordoma — Another area from the same tumor to show large multi-nucleated "physaliphorous" cells and nononuclear tumor, surrounded by same fibrillar matrix. MGG x 400

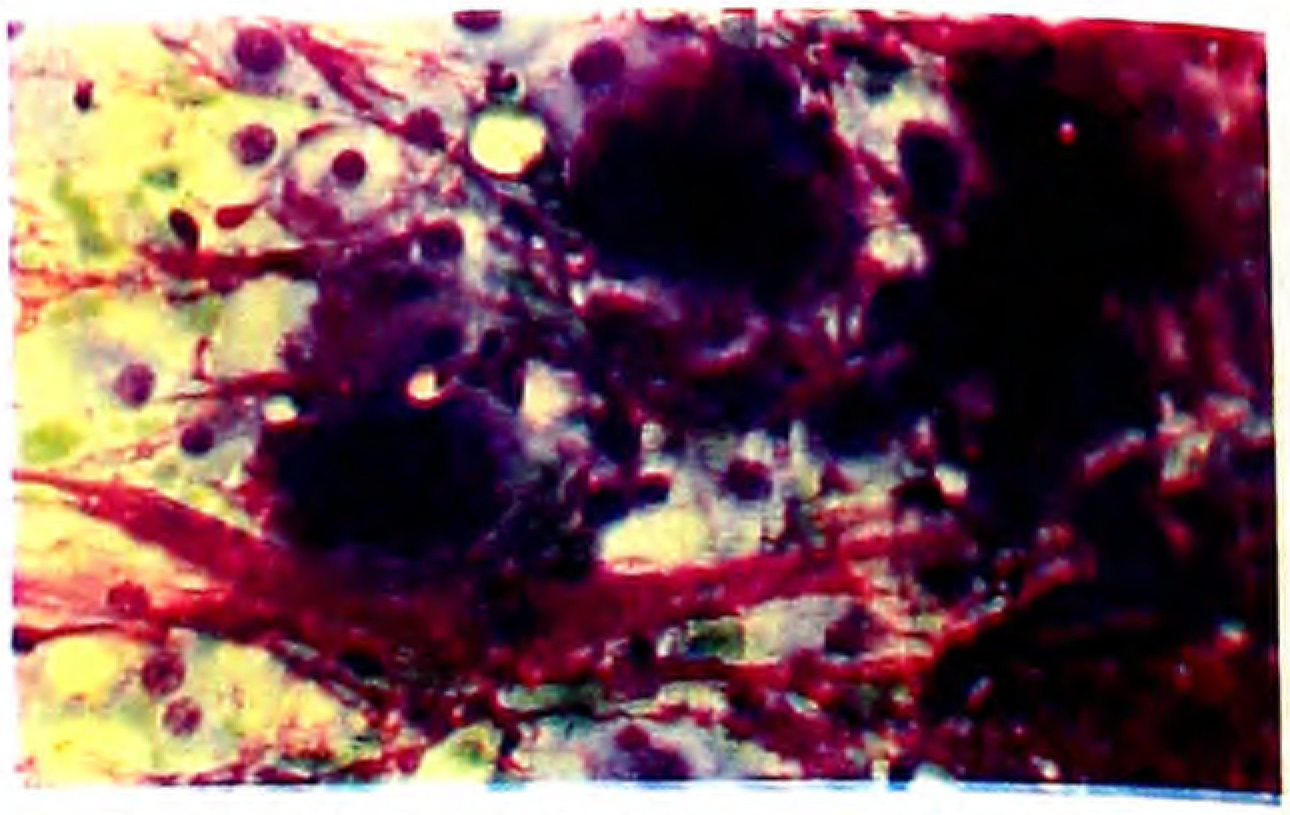

Fig. 16. FNAC, Adamantinoma — tibia show large dense clusters and ribbons of basaloid tumor cells. Note clear background with no malignant diathesis. MGG x 200

so called "physaliphorous" cells. No mitotic figures or necrosis was seen. The smears with clinical and x-ray findings were consistent with the diagnosis of chordoma. Histopathology confirmed the smear diagnosis of chordoma.

Adamantinoma

One case of adamantinoma on FNAC was recorded in lower diaphysis tibia in a 40 year male in 1994 (Table 7). This was a recurrent tumor diagnosed as adamantinoma on excision biopsy, by the author in 1993. X-ray showed a radio-lucent area with sclerotic areas involving the cortex primarily extending into the medulla. There was no break in cortex, but cortex was so thin that it could be penetrated easily by a 22 guage needle.

Cytologic Findings:

Smears were highly cellular with multiple large clusters of cells, round to spindle with round uniform nuclei, fine chromation & scanty ill defined cytoplasm, almost forming syncytium, mimicking basaloid squamous cell carcinoma. No mitotic figures or necrosis were present. This was a bland epithelial tumor with no stroma at all. With the previous knowledge of the histopathology, smears were consistent with adamantinoma (Fig.16).

Discussions:

FNAC is now widely accepted and applied diagnostic procedure. In spite of attempts at bone aspiration in early 1930's[1,2] this area has not kept pace with other sites. This may be partly explained by the non-familiarity of cytopathologists with pathology of bone tumors. Lytic bone lesions for suspected metastasis, however have been the target of aspiration for many decades. With management of some primary bone malignancies like osteosarcoma & Ewing's sarcoma by preoperative chemotherapy, coupled with advantages of FNAC over open & needle biopsy, the FNAC of primary bone tumors is now gaining more attention[5-25]. It is essential to remember that FNAC diagnosis of bone tumors like histopathologic diagnosis requires complete evaluation of clinical & radiological data. It is also true that malignant tumors are diagnosed more correctly than benign ones.

A review of FNAC records on bone tumors by the author over last 11½ years (Jan 1991-June 2002) revealed 110 bone tumors. It is the FNAC pattern of primary malignant bone tumors which will be discussed here.

Myeloma (MM):

The plasma cell tumors of bone were the most common, with a total of 20, 12 of which were aspirated from sternal site & the remaining 8 from the non-sternal sites (Table 2). The smear pattern in these two groups were similar, with the difference that clusters of tumor cells were also seen in FNAC smears. Both sternal & non-sternal aspirates showed dispersed cells of mature and immature plasma cells with crowding out of normal haemopoietic elements (Fig.1-3)

Myeloma is the most common primary bone malignancy, but it is generally taken care of by hematologists. There were 12 such cases in this study which after sternal aspirate diagnosis were investigated further by haematologists. Cytopathologists occasinally get an opportunity to aspirate such tumors that present as solitary lytic bone lesions. Such eight cases in this study were subjected to incisional biopsy which confirmed the diagnosis of myeloma. Such reports are available in the literature.[2,8,16,24,25]

Diagnosis of plasma cell tumors is generally straight-forward; but other lesions like chronic osteomyelitis & immunoblastic lymphomas need consideration. In chronic osteomyelitis plasma cells are invariably accompanied by acute inflammatory cells. The poorly differentiated myelomas will show a range of cells with plasmablasts and mature plasma cells in contrast to immunoblastic lymphoma. In difficult cases immunocyto chemistry will be required.

Osteosarcoma (O.S):

Out of 12 cases of osteosarcoma, 11 were diagnosed on FNAC (Table-3). One case did not yield any material. Smears in these cases showed pleomorphic sarcoma as described earlier. The stroma compatible with osteoid in variable amounts was seen in all the 11 cases. Thus the cytologic picture supported by clinical & radiologic picture was compatible with the diagnosis of osteosarcoma.

The diagnositic accuracy of osteo-sarcoma in FNAC depends on the type of the lesion. (osteolytic vs osteosclerotic) & experience of the cytopathologist with bone pathology. In once study[26], out of 56 cases of O.S. correct diagnosis was made in 45 cases (80.4%). In another study[24], of 55 cases a definite diagnosis was made only in 19 cases (32.2%), the material in the remaining cases was either inadequate or not present at all. Some authors have attempted morphologic grouping of O.S. with an accuracy of 80.4%[26] and 87.3%[27] This is only of academic importance and does not have any bearing on the management of the patient.

Presence of osteoid, unlike histopathology is not mandatory for the diagnosis of O.S. in smears. Osteoid is indistinguishable from hyalinised collagen, but a bright or faint eosinphilic material, enclosing tumor cells or lying dispersed in the background in a pleomorphic sarcoma with appropriate clinical and radiological data, is compatible with osteoid. Thus osteoid has been reported in 35%[26] to 40%[28] of the cases. However, the author has been able to find some stroma compatiable with osteoid in all the 11 cases.

The differential diagnosis of O.S in smears would include pleomorphic MFH, pleomorphic metastatic carcinoma, Giant cell tumor and Ewing' sarcoma depending upon the morphology of osteosarcoma. Careful study of cytology with clinical and radiologic details should help to arrive at the diagnosis even without the ancillary studies in most of the cases.

Chondro-sarcoma (SC)

This study included 4 cases of chondro-sarcoma out of 15 chondroid neoplasms (Table-4). In three cases smear were suggestive of aggressive chondroid neoplasm and in the fourth case as benign chondroma. Radiographs helped to suggest the diagnosis of low grade chondrosarcoma in case 4.

There are limited reports of FNAC on the chondrosarcoma of bone with general acknowledgement of limitation of FNAC diagnosis of these tumors [12,14,25,29,30]

In FNAC smears the qualitative and quantitative differences in the cellularity, nuclear morphology including size, chromatin pattern, presence and number of nucleoli and number of nuclei as well as mitosis are the basis of tumor grading as in histopathology. However the permeative growth pattern appreciated in histopathology is not available in smears. Thus grade I chondro-sarcoma shows a myxohyaline stroma with cellular paucity, comprising of chondrocytes with a low N/C ratio. But these are difficult to diagnose as chondrosarcomas on FNAC without a clinical and radiographic support as exemplified by the fourth case in this study Grade 2 smears are more cellular, the tumor cells lying both dispersed and in fragments show mononucleated cells with nuclear atypia lying in abundant chondroid matrix. Thus there were three such cases in the study where smears were suggestive of aggressive chondosarcoma supported by radiographs and confirmed by histopathology as chondosarcoma grade 2. The smears of grade 3 chondosarcoma show richer cellularity and more definite nuclear atypia with chondroid stroms. The poorly differentiated and de-deffernitiated tumors are difficult to diagnose as chondosarcomas and are best diagnosed as malignant mesenchymal tumors. Author has experience of one such soft tissue chondosarcoma on FNAC which was diagnosed as soft tissue sarcoma NOS. (Unpublished)

The differential diagnosis of chondro-sarcoma should include chondroma, chondromyxoid fibroma and chondroblastic osteosarcoma. The clinical picture and radiology should be taken in to consideration to arrive at the diagnosis.

Non-Hodgkin's Lymphoma (NHL)

Six cases of non-Hodgkin's lymphoma of bone were encountered in this study with age range of 30-70 years, including 4 males and two females with 4 in flat bones and 2 in long bones (Table-6). Investigations later proved only 2 cases limited to bone. The remaining 4 cases were disseminated but had presented primarily as bone lesions. Criteria for the diagnosis of lymphoma in smears were same as in the lymph nods[3, 4]. Using Working Formulation, 4 cases were categorized as large cell and 2 cases as mixed cells lymphoma. Histopathology available in all the 6 cases confirmed these as aggressive lymphoms (Table-6)

There are a few reports of FNAC of bone lymphomas in children and adult[25, 31].

Primary non-Hodgkin's lymphoma of bone is rare. It constituted 8% of malignant bone tumors in Mayo clinic[32]. It may occur at any age and site, the most common sites being the bones with persistent marrow[38].

The differential diagnosis of bone lymphoma includes chronic osteomyelitis Ewing's sarcoma, metastatic neuroblastoma, metastatic rhabydomyo sarcoma and metastatic anaplastic carcinoma depending upon the age.

Ewing's Sarcoma (E.S)-PNET

Ten cases of Ewing's Sarcoma (E.S)-PNET were recorded with age range of 4½ to 23 years including 8 males and 2 female, with 9 cases from flat bones and 1 from long bone (Table 5). Smear pattern described above was that of round cell tumor, without stroma. The tumor with frequent rosettes are labeled as Ewing's Sarcoma (E.S)/ PNET. There was one such case in this study.

E.S-PNET are malignant round cell tumors of bone and soft tissue, of neuro-ectodermal nature[33]. E.S represents the undifferentiated end and PNET the differentiated end of the spectrum, without significant biologic or therapeutic considerations.

The cytology of E.S. has been described in details by various authors[34-37]. The diagnostic accuracy generally has been high except one study[37].

The differential diagnosis includes non-Hodgkin's lymphoma, neuroblastoma, small cell osteo-sarcoma and metastatic rhabdomyosarcoma. The smears offer better morphology than histopathology and therefore in typical cases, there is no difficulty in making the diagnosis as in the present study. The atypical cases however, would require cell makers, cytogenetics and molecular studies[38].

Chordoma

One case of chordoma was diagnosed in a 40 year male in the sacrum (Table 7). Smears showed abundant magenta coloured mucoid material fibrillary and bubbly with round uniform cells scattered with 1-2 vesicular nuclei and vacuolated cytoplasm. Occasional multinucleiated physalliphorous cells were also present.

Chordoma are rare tumors of bone[32] derived from notochord. 85-90% are seen at distal and proximal ends of the vertebral column. The mean age of on-set of sacaral chordoma is 35 years[32]. The tumors are locally destructive with rare metastasis. FNAC reports of this tumor are virtually all from the sacro-coccygeal region[15, 25, 26; 39-44] The smears diagnosis is generally straight-forward. However, other diagnostic possibilities like metastatic mucinous adeno-carcinoma, myxiod chondrosarcoma have to be ruled out. The physaliphourous cells are not essential for diagnosis[43] In problem cases, immunocytochemistry should help. Staining for epithehil markers and S-100 protein would help to differentiate them.

Adamantinoma

One case if adamantinoma was reported on FNAC in a 40 male in lower diaphysis of tibia (Table 7). This was a recurrent tumor diagnosed as adamantinima by the author on histopathology, one year earlier in 1993. Smears were hypercellular with cohesive clusters of basaloid squamous cells with peripheral palisading. The tumor cells were monomorphic uniform nuclei, had fine chromatin and ill defined cytoplasm. Stroma was absent. There were no mitotic figures or necrosis FNAC diagnosis of the abdamantinoma was possible only with the previous knowledge of histopathology.

Admantinoma of bone is a rare neoplasm representing 0.3% - 1% of malignant bone tumors[32, 38]. Tumor is slightly more common in males with age ranges from 4-72 years and 90% involve the tibia[38]. There are a few reports of FNAC on admantinoma of the bone[45, 46].

References

1. Martin HE, Ellis EB, Biopsy by needle puncture and aspirates. Ann Surg 1930;92 169-181.
2. Coley BL, Sharp GS, Ellis EB, Diagnosis of bone tumors by aspiration. Ann J.Surg 1931;13: 215-224.
3. Heerde Van P. Lymph nodes. In: Mannual of Aspiration Cytology. Eds Orell SR, Sterref GF, Walters MN, Whitaker D. Third Edition. Churchill Livingstone 1999, 74-108.
4. Khan AR, Omer S, Rashid S, Besina Syed. Utility of Fine needle Aspiration in the diagnosis of the nodal primary non-Hodgkin's lymphoma. JK Pract 2002, 3; 197-204.
5. Synder RE, Coley BL. Further Studies on diagnosis of bone tumors by aspiration biopsy. Surg Gynacol Obster 1945;80: 517-522.
6. Stormby N Akerman Cytodiagnosis of bone lesion by means of fine needle aspiration biopsy. Acta Cytol 1973, 17:166-

172.

7. Agarwal PK, Wahal KM, Cytopathologic study primary tumors of bone and joints. Acta Cytol 1983, 27; 23-27.
8. El-Khoury GV, Ferepka RH, Mickelson MR. Fine needle aspiration of bone. J Bone Joint Surg. 1983, 65(A): 522-525.
9. James LP. Frable WJ. Fine needle aspiration of bone lesions (Abstr). Acta Cytol 1983, 27:559.
10. Feldman PS, Covell JL. Cyto Diagnosis of bone and soft tissue lesions by fine-needle aspiration (Abstr). Acta Cytol. 1983, 27; 558.
11. Xiaojing P. Xiangchen Y. Cytodiagnosis of bone tumors by fine needle aspiration. Acta Cytol 1985, 29; 570-575.
12. Lay field LJ, Alasgow BJ, Anders KH, Mirra JM; Fine needle aspiration cytology of primary bone lesions. Acta Cytol 1987, 31; 177-184.
13. Ashok Raj G. Bhatia A; Fine needle aspiration cytology of bone tumors, Indian J. Cancer 1989, 26;260-264.
14. Kumar RV, Rao CR, Hazarika D, Mukerji G, Gouda BM. Aspiration Biopsy, Cytology of Primary Bone lesions. Acta Cytol. 1993, 37;83-89.
15. Nanda M. Rao ES, Behera KC, Dao S Mohanty L; Fine Needle Aspiration Cytology in malignant bone tumors. Indian J. Pathol Microbiol 1994; 247-253.
16. Mondal A; Fine Needle Aspiration Biopsy Cytology in the diagnosis of Osteolytic lesions of bone. J. Indian Med. Assoc 1994; 92:118-119.
17. Mandal SC, Mukerji M; Cytopathologic study of bone tumors. J. Indian Med. Assoc 1994; 92: 338-346.
18. Roy M. Battacharaya A. Sanyal S. Roy R. Das Gupta S. Fine needle aspiration cytology of bone and joint lesions: an assessement. J. Indian Med Assoc 1995, 93:259-262.
19. Agarwal PK, Goel MM, Chandra T. Agarwal S. Predictive value of fine needle aspiration cytology in bone lesions. Acta Cytol 1997, 41:659-665.
20. Boomer K, Ramzy I, Mody D; Fine needle aspiration biopsy in the diagnosis and management of bone lesions. A study of 450 cases. Cancer cytopathol 1997, 81;148-156.
21. Jorda M, Rey L, Hanly A, Ganjel Azar. Fine needle aspiration cytology of bone — Accuracy and pit fall of Cytodiagnosis Cancer Cytopathol. 2000, 90; 47-54.
22. Wedin R, Baner HC, Lambert S, Soderlund V, Tani E; Cytologic diagnosis of skeletal lesions. Fine needle aspiration in 110 tumors. J. Bone Joint Surg 2000, 82B, 673-678.
23. Jambhekar NA, Moonim MT, Khatib Y. Bone FNAC - Expectations and limitations: A review - Part I J of Cytol 2001, 18(1): 1-7.
24. Jambhekar NA, Moonim MT, Khatib Y. Bone FNAC - Expectations and limitations: A review — Part II J. of Cytol 2001, 18(2): 57-72.
25. Pai MR, Coimbatore RV, Naik R, Lugade M. Role of fine needle aspiration cytology in the diagnosis of bone tumors J. of Cytol, 2002; 19(1); 33-39.
26. Tune M, Ekinci C, Chondrosarooma diagnosed by fine needle aspiration cytology Acta Cytol 1994, 40: 283-288.
27. White VA, Fannuing CV, Ayala AG: Osteosarcoma and the role of fine needle aspiration — A study of 51 cases. Cancer 1988, 62; 1238-1246.
28. Bhatia A, Ashok Raj G: Cytologic diversity osteosarcoma. Indian J. Cancer 1992, 29; 56-60.
29. Ward WG Sr. Kilpatrich S: Fine needle aspiration of primary bone tumors Clin orthop. 2000, 373;80-87.
30. Akerman M, Berg NO, Oresson BBBBM: Fine needle aspiration biopsy in the evaluation of tumor - like lesions of bone. Acta ortho Scand 1976; 47; 129-136.
31. Nieimann TH, Thomas PA, Primary lymphoma of bone — diagnosis by fine needle aspiration biopsy in a paediatric patient. Diagn. Cyto pathol. 1995, 12; 165-167.
32. Uoni KK, Inwards CY: Tumors of the osteo articular system. In Diagnostic Histopathology of Tumors, Ed. Christopher DM, Flectcher. II Edition Churchill Living stone 2000, 1541-1606.
33. Ushigoine S, Shimoda T, Nikaido T. Primitive neuro-octodermal tumors of bone and soft tissue: with reference to histologic differentiation in primary or metastatic foci. Acta Pathol 1989, 42;483-492.
34. Fanning TV, Katz RL, Ayala AG. Fine needle aspiration of Ewing's Sarcomawe of bone — a study of 57 specimens Acta Cytol 1985, 29; 932.
35. Mondal A, Misra DK; Ewings Sarcoma of bone: a study of 71 cases by fine needle aspiration cytology. J. Indian Med Assoc 1996, 94: 135-137.
36. Sahu K, Pai RP, Khadikar UN: Fine needle aspiration cytology of Ewing's sarcoma family of tumors. Acta cytol 2000, 44: 332-336.
37. Dupay DE. Rosenberg AE, Punyara TA Bandhu Tan MH, Mankin Ha. Accuracy of CT-guided needle biopsy of musculoskeletal neoplasms. Am J. Radiol 1998, 171; 759-762.
38. Schajowicz F. Tumor and tumor - like lesions of bone. 2nd ed. Springer-Verlag 1994, 459-467.
39. Dahlni DC Unne KK. Bone tumors general aspect and data 8542 cases. 1986. Charles C. Thomas, Springfield IL.
tumors by aspiration biopsy. Surg Gynae Obstel - 1954, 80; 517-522.
40. O Dowd G, Schumann GB, Aspiration Cytology and cytochemistry of coceygial chordoms. A case report and review of literature. Acta Cytol 1983, 27: 187.
41. Nighawan VS, Rajwanshi A. Das A: Fine needle aspiration cytology of sarco coceygial chordoma diagn cytopathol 1998, 4: 144-147.
42. Lafer C, Rosia RP. The cytology of chordoma Acta Cytol, 1978; 22: 53-55.
43. Thompson SK, Callery RT. Cytologic diagnosis of a chordoma without physaliphorous cells. Diagn, Cytopathol, 1988; 4: 114-117.
44. Wallas L, Kind blome G: fine needle aspiration biopsy in the pre-operative diagnosis of chordomas-a study of 17 cases with the application of electronmicroscopic, histochemical and immunocyto chemical examination. Human Pathol,1991,22:53-28.
45. Galera- Davidson H, Fernaudez-Rodriguez A, Torres-Olivera FJ, Gonuz- pascual A, Morenofernandez A: Cytological diagnosis of recurrent adamantinoma. acta Cytol 1989, 33:635-638.
46. Laucirica R, Mody D, macleay L, Kearus R,1: Adamantinoma- A case repoart with aspiration Cytology and differential diagnosis and immuno histo hemical correlation acta Cytol 1992, 36:951-956.

INFECTIONS IN PREGNANCY

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I. INTRODUCTION

Viral infections during pregnancy can occur in utero (i.e. congenital infections), at birth, or in the neonatal period, and account for 2-3% of congenital anomalies. Each type of viral infection may have a different outcome depending on:

1. the timing of the exposure,
2. the gestational age of the fetus,
3. the level of inoculum below which exposure does not result in teratogenesis,
4. whether this is a primary (first experience) infection
5. whether this is a secondary (recurrent) infection
6. or whether this is a latent infection.

II. COMMON VIRAL INFECTIONS DURING PREGNANCY

A. CYTOMEGALOVIRUS

Human Cytomegalovirus (CMV), the most common congenital infection in neonates in the United States, infects people of all age groups, socioeconomic backgrounds, and geographic regions. It is a DNA virus and belongs to the herpesvirus group. The presence of maternal humoral antibody does not confer fetal protection in subsequent reinfection or reactivation as seen with other viruses. CMV has been cultured from virtually all body fluids, including saliva, semen, and cervicovaginal secretions.

CMV can be transmitted through intimate contact with another person shedding the virus, organ or bone marrow transplantation, blood transfusion from CMV-seropositive donors, and vertical transmission. *An ascending route of infection has been postulated but is unlikely with intact amniotic membranes.*

CMV Management

Currently there are no pharmacological regimens available for the treatment of maternal and fetal CMV infections. (Ganciclovir is only approved for CMV retinitis in patients with AIDS). Reports of placental transfer of Ganciclovir and neonatal treatment with hyperimmune gamma globulin have shown promise, however, the data are lacking.

The best long-term answer may be the live attenuated CMV vaccine (Towne 125 strain). Reports indicate that it is safe, provides some protection, and offers an economical alternative. Concerns about the vaccine include its potential for reactivation (reinfecting), viral shedding in breast milk and the cervix, and oncogenesis.

CMV Current Recommendations

Routine serologic CMV testing during pregnancy is not recommended. Individuals involved in childcare and patients with young children are potentially at greater risk. It is important these individuals are counseled to practice safe handling techniques, frequent hand washing and proper use of latex gloves when exposed to any bodily secretions.[1]

B. Varicella-Zoster Virus

Varicella-Zoster Virus (VZV), a DNA virus that belongs to the herpesvirus group is the causative agent of chickenpox and shingles. Varicella represents primary infection, whereas shingles represents reactivation of a latent virus located in the dorsal root ganglia. Varicella is highly communicable. About 2% of Varicella cases occur in adults of reproductive age and this group accounts for 45% to 55% of Varicella-related deaths. One to five cases in 10,000 pregnancies are estimated to occur.

VZV usually is transmitted by infected secretions from the nasopharyngeal regions. Once inoculation has occurred, primary viremia (viral replication) takes place for four to seven days followed by a secondary viremia (exanthem) at approximately day 14. Patients are considered infectious 24 to 48 hours before the rash, and remain infectious until crusting of the exanthem occurs. Primary varicella infection generally confers lifelong immunity; however, a few cases of reinfection have been reported.

VZV Management

Treatment of VZV with acyclovir has proven beneficial for the mother if instituted within 24 hours of the viral exanthem. Oral acyclovir (safe during pregnancy, class C) reduces the duration and numbers of lesions and improves the associated constitutional symptoms.

Varicella pneumonia should be treated with intravenous (IV) acyclovir. Maternal treatment with acyclovir, regardless of route of administration, has not been shown to prevent or buffer the fetal effects of congenital varicella infection. The use of other antibiotics in conjunction with acyclovir to prevent superimposed bacterial respiratory infections is controversial.

VZV Current Recommendations

Nonpregnant women of reproductive age should be questioned concerning previous varicella infections.

1. No history of varicella /or if a nonimmune status is documented: Patients should be offered the varicella vaccine with recommendations of delaying conception for at least 3 months.
2. Women who do not recall a past varicella infection: Antenatal VZV antibody determination in women with a negative history is controversial due to the high cost and low yield, but many practitioners obtain this test at the first prenatal visit and 80% to 90% have antibodies.
3. Pregnant patients with negative or indeterminate histories: should avoid persons who are infectious. If exposure occurs, prophylactic administration with Varicella-Zoster immunoglobulin (VZIG) should be given to attenuate and perhaps prevent varicella if the patient is VZV-antibody negative. VZIG should be given within 96 hours of exposure.

The administration of maternal VZIG does not prevent or ameliorate congenital varicella infection. Neonates whose mothers develop varicella 5 days before delivery or 2 days following delivery should receiveVZIG after birth.[2-7]

C. Parvovirus B19

The parvoviruses are small single-stranded DNA viruses caused by erythema infectiosum (EI). Viruses are transmitted through respiratory secretions and hand-to-mouth contact. The patient is generally infectious one week after exposure and becomes noninfectious with the onset of the rash. Fifteen to 25% of infected patients are asymptomatic, most common symptoms include a reticular, lace-like rash on the trunk and arthralgias.

As seen with CMV, there seems to be occupational risks for infection for childcare workers and teachers. When parvovirus B19 outbreaks occur, options for preventing transmission are limited.

B19 Management

Fetal intrauterine vascular transfusion (IVT) is indicated when anemia with hydrops or reticulocytopenia with anemia are present[8,9].

D. Viral Hepatitis in Pregnancy Acute and chronic necro-inflammatory liver disease caused by hepatotropic virus infection often affects women of child-bearing age and their infants. In the United States, end-stage liver disease caused by chronic hepatitis C virus (HCV) is the most common indication for orthotopic liver transplantation (OLT). HCV infection often is acquired by teenagers and young adults as a result of drug use and also is common in persons with a history of sexual promiscuity. Thus, individuals providing health care to young women are likely to encounter patients with viral hepatitis and must be prepared to deal with issues related to acute and chronic infection, sexual and vertical transmission, and screening and treatment

Clinical Features of Viral Hepatitis

The clinical features of each type of viral hepatitis are similar and almost exclusively the consequences of liver cell injury; on rare occasions, individuals with viral hepatitis will have extrahepatic (immunologic) manifestations of their disease. The presentation of hepatitis in pregnant women, as a rule, is no different from that in nonpregnant individuals and is determined by the severity of the liver damage and whether or not it is acute or chronic. Most individuals who are exposed to a hepatitis virus do not develop clinically apparent disease. For this reason, many people are unaware that they have been infected with a hepatitis virus and are surprised if serologic testing demonstrates hepatitis virus antigens or antibodies.

Table 1. Acute viral hepatitis in the U.S.

HAV	HBV	HCV	HDV	HEV
32%	42%	20%	2%	1%

Persons with clinical viral hepatitis initially have nonspecific complaints, including fatigue, malaise,

Table 2. Classification of the hepatotropic viruses

Species	HAV	HBV	HCV	HDV	HEV
transmission					
Incubation period	15-50d	40-150d	14-140d	20-45d	15-65 d
Clinical illness	Pediatric 50% adult, 80%	30%	5%	10%	80%
Chronicity	No	5%	85%	Yes	No
Fulminant course	<1%	<1%	Rare or no	<5%	Pregnancy
Antigen test	Fecal	Serum	None	Serum	Fecal
Antibody test	Serum	Serum	Serum	Serum	Serum
Nucleic acid test	Feces/serum	Serum	Serum	Serum	Feces
Vaccine	Yes	Yes	No	Yes (HBV)	No
IG	Yes	Yes	No	No	No

anorexia, nausea, headache, and myalgias, possibly associated with low-grade fever. It may be mistaken for a flu-like viral syndrome or even for the normal physiologic effects of pregnancy itself; otherwise, jaundice develops within 2 to 10 days of the onset of symptoms.

Chronic viral hepatitis is asymptomatic either indefinitely or until there is sufficient liver damage for the patient to develop manifestations of end-stage liver disease. Some persons experience periodic clinical exacerbations that resemble mild, moderate, or even severe acute hepatitis. Many cases of chronic viral hepatitis, therefore, are diagnosed after serum aminotransferase levels incidentally are noted to be abnormal. This commonly occurs when an apparently healthy young woman becomes pregnant and consults an obstetrician. Physical examination also may be normal or the patient may have subtle physical findings consistent with early cirrhosis, including palmer erythema, splenomegaly, and a small liver. Ultimately, the natural history of viral hepatitis in a given patient is determined by the particular agent causing the infection and the host response to infection[10-13] (Tables 2 & 3).

Table 3. Serological diagnosis of acute viral hepatitis

Disease	Serology	Comments
Hepatitis A	IgM anti-HAV	Specific
Hepatitis B	HbsAg	May be negative late
	IgM anti-HBc	Usually indicates acute Hepatitis B
Hepatitis C	Anti-HCV	Often appears late: 4-8 weeks
Hepatitis D	HbsAg and Anti-HDV	Anti-HDV may appear late
	(+) IgM anti-HBc	Coinfection
	(-)IgM anti-HBc	Superinfection
Hepatitis E	All negative	Travel history
Hepatitis (Non-A-E)	All negative	History of risk factors

1. Hepatitis A Virus

HAV infection is no severer in pregnant women than in nonpregnant individuals. Intrauterine and perinatal maternal-fetal transmission of HAV have been reported, however, maternal HAV infection during pregnancy is not associated with fetal loss or developmental abnormalities.

Passive immunization with pooled human serum immunoglobulin (IG) containing anti-HAV IgG three to six months before, or within two weeks of exposure to HAV prevents or attenuates hepatitis in 85% to 95% of treated individuals. The IG and HAV vaccine both are safe for use in pregnant women.

2. Hepatitis B Virus

Acute and chronic HBV infection are diagnosed by a positive serum HBV surface antigen (HBsAg) test. Recent infection is indicated by the presence in serum of IgM antibody to HBV core antigen (anti-HBc), whereas predominant serum IgG anti-HBc is found in persons with chronic infection. Active viral replication in acute or chronic HBV infection is seen in patients with serum HBV e antigen (HBeAg). In contrast, serum antibody to HBeAg (anti-HBe) is found in individuals recovering from acute HBV infection and also in persons with chronic HBV infection without active viral replication. HBV infection is no severer in pregnant women than in nonpregnant individuals, unless there is also severe chronic hepatitis or secondary cirrhosis and associated complications in a chronic HBV.

Table 4. Serological Diagnosis of Chronic Viral hepatitis

Disease	Serology	Confirmation
Hepatitis B	HbsAg –Active viral replication (HbeAg, HBV DNA) –Low/absent viral replication (anti-HBe, (-) HBV DNA)	(-) IgM anti-HBc
Hepatitis C	Anti-HCV (EIA) Anti-HCV	(±RIBA)HCV RNA
Hepatitis D	HbsAg and anti-HDV (>1:100)	HDV antigen
Hepatitis Non-B-D	All negative	History of risk factors, exclusion of other diagnosis

Pregnant women suspected of having been exposed to HBV should be treated with HBIG and innoculated with HBV vaccine. This therapy is safe and effective during pregnancy.

3. Hepatitis C Virus

Acute infection almost always is subclinical and becomes chronic in over 85% of cases. Chronic HCV infection predisposes affected individuals to develop cirrhosis, end-stage liver disease and HCC. In addition, chronic HCV infection is associated with a number of extra-hepatic manifestations including arthritis, keratoconjunctivitis sicca, lichen planus, porphyria cutanea tarda, and essential mixed cryoglobulinemia, with or without glomerulonephritis. These disorders, presumably, may be diagnosed in pregnant women.

In the United States, the highest prevalence of HCV infection is in persons between the ages of 30 and 49 years (3% to 4%). This disease, therefore, is not uncommon in women of childbearing age. There is no evidence that pregnancy alters the natural history of HCV or that HCV interferes with normal pregnancy, unless the patient has cirrhosis with associated complications. HCV, however, may be transmitted vertically when maternal viral titers are unusually high, for example, in persons with HCV and HIV Coinfection. Maternal serum HCV RNA levels of greater than or equal to 10^{19} copies per ml have been reported to be associated with vertical transmission in as many as 36% of cases.

4. **Hepatitis D Virus Hepatitis D** (delta) virus (HDV) infection may be acute or chronic, and does not occur in the absence of acute or chronic HBV infection because HDV requires HBV for coating and cell-to-cell spread. Acute HDV is diagnosed by detection of the serum anti-HDV IgM antibody in a patient who also has a positive serum HBsAg test. While on the contrary, persons with chronic HDV and HBV infections have serum anti-HDV IgG antibody titers of greater than 1 to 1000 and positive serum HBsAg tests.

   Most individuals with chronic HDV have hemophilia or a history of IVDU. Perinatal transmission of HDV is rare, and there are no documented cases of vertical transmission of HDV in the United States.

5. **Hepatitis E Virus**

   HEV is a disease of people living in the third world countries, usually occurring in widespread outbreaks, and caused by fecal contamination of drinking water. In Western, industrial nations, HEV infection is rare and has been diagnosed exclusively in travelers returning from Third World countries. In nonpregnant patients, HEV infection is fulminant in less than 1% of patients, whereas in pregnant women, it is fulminant in as many as 20% of patients. Similarly, the overall fatality rate of HEV is 0.5% to 4%. However, during pregnancy in the first trimester, the fatality rate is 1.5%. It is 8.5% during the second trimester, and 21% during the third trimester. It is unknown why this virus causes especially severe disease in pregnant women.

   HEV infection during the third trimester of pregnancy is associated with an increased number of fetal complications. The death of neonates caused by HEV infection is much more common than it is from other types of viral hepatitis. HEV infection is never chronic.

   Prevention of HEV epidemics depends on improvements in sanitation and protection of the purity of water supplies. Pregnant women should not travel to areas where there are known cases of HEV because immunoprophylaxis against HEV infection is not available.

6. **Hepatitis F, G and X Viruses**

   It appears that 5% to 20% of cases of acute and chronic hepatitis are not caused by known hepatitis viruses, or toxic, metabolic or immune-mediated mechanisms. Untyped hepatitis viruses may infect women during pregnancy and be a source of diagnostic confusion, particularly during the third trimester, when jaundice may be a complication of pregnancy-induced hypertension or a sign of acute fatty liver of pregnancy.

   III. Toxoplasmosis Toxoplasmosis is caused by *Toxoplasma gondii*, an obligate intracellular parasite. It is a significant human pathogen that can affect the fetus, neonate, adolescent, and adult, particularly the immunocompromised adult.

Transmission to humans occurs by ingestion of oocytes or cysts from animals or contaminated soil. Undercooked meats such as pork or beef are sources of human infection. Infection also can occur by insect contamination of food, contact with oocytes from the feces of infected cats, or contact with other infected materials. The domestic cat is the only definite host currently identified.

Primary infections occur in approximately 0.6% of reproductive-age women. Approximately 40% of pregnant women in the Unites States have serologic evidence of previous *T. gondii* infection and are not at risk for delivering a fetus with congenital *infection*. Primary infection accounts for 3000 infants delivered annually most of these infants are asymptomatic at birth but develop sequelae later in life.

Neonatal Infection

Treatment

In the case of an acute maternal infection, immediate treatment is warranted. It seems that treatment reduces, but does not eliminate, the risk for congenital infection. Spiramycin may reduce vertical transmission by 50% to 60%. Once serologic evidence of infection has been confirmed, Spiramycin is available only through the US Food and Drug Administration (FDA). Spiramycin is not recommended for the pregnant patient at risk unless fetal infection is suspected. Pyrimethamine, sulfonamides, and folinic acid are added to spiramycin because they eradicate maternal and fetal parasitemia when compared with spiramycin alone. Treatment can result in successful pregnancy outcomes.

Recommendations

1. Routine screening during pregnancy is not recommended in the United States, except in those patients with AIDS.
2. Routine serologic screening during pregnancy is probably advantageous in areas where there is a high frequency of seropositivity.
3. Patient education in avoiding undercooked meats, use of gloves when working with soil, and avoidance of caring for outdoor cats[14-16]

IV. Tuberculosis and Pregnancy

Tuberculosis remains an important respiratory disease. Recent epidemiological data suggest an increasing worldwide prevalence of tuberculosis. Although approximately 1% to 2% of all pregnant women in the United States exhibit tuberculin skin test reactivity, less than 10% have active tuberculosis lesions. Pregnant women with tuberculosis are usually symptomatic with cough (74%), weight loss (41%), fever (30%), fatigue (30%), and hemoptysis (19%). Pregnancy does not seem to affect the clinical course of tuberculosis. Hematogenous spread of *Mycobacterium tuberculosis* may result in congenital tuberculosis. Given the complex nature of treatment during pregnancy and the development of multidrug-resistant strains of tuberculosis, treatment should be individualized in consultation with infectious disease services.

Mantoux Skin Test

Screening of all pregnant women with the Mantoux skin test which involves intradermal injection of 0.1 ml of 5 TU (Tuberculin Unit) strength PPD (Purified Protein Derivative) identifies most of the those with inactive, healed, prior infection and those with true symptomatic and asymptomatic disease.

Despite the generalized decrease in cell-mediated immunity, the tuberculin skin test is frequently positive in pregnant women, and studies have demonstrated no effect of pregnancy on cutaneous delayed hypersensitivity to tuberculin.

High-risk groups include: Foreign-born people, health care workers, residents of nursing homes, the homeless, drug addicts, and those residing in a high TB prevalence[17]

Table 5. Interpretation of Skin Tests for Tuberculosis (TB*)

Area of Induration (mm)	Reading
< 5mm	Negative
≥ 5mm	Positive if patient is a known TB contact, has abnormal CXR, clinical evidence of TB or HIV positive
≥10 mm	Positive in any high risk group or high prevalence group
≥15mm	Positive

V. Asymptomatic Bacteriuria: All pregnant women should bescrened with a urine culture at their fist prenatal visit; those without symptoms of UTI who grow more than 25,000 organisms/mL of urine have asymptomatic bacteriuria. This occurs in 2% to 7% of pregnancies, with an increased risk among those with previous UTIs, low socioeconomic class, diabetes, or sickle cell trait. Women with asymptomatic bacteriuria have a much greater risk up to 28% of progressing to pyelonephritis, which can pose risks to mothers and fetuses. Although most isolates are from the enterobacteriaceae, such as *Escherichia coli* or *Klebsiella* sp., group B streptococci are also common.

Diagnosis

Based on culture of clean-catch urine at the initial prenatal visit, asymptomatic bacteriuria is diagnosed if more than 25,000 organisms/mL are found. Lower concentrations may be clinically relevant if a patient is symptomatic or taking antibiotics.

Treatment

Although the culture results can be used to obtain organism sensitivities, most patients are treated empirically with oral antibiotics. Appropriate choices include ampicillin, 500 mg three times a day; nitrofurantoin, 100 mg twice a day; or cephalexin, 250 mg to 500 mg, three times a day; each for 7-10 days. Local resistance patterns and patient allergies may limit the initial choices.

Prevention

The incidence of acute pyelonephritis is significantly decreased in patients treated for asymptomatic bacteriuria, but one third of treated women have persistence or recurrence of bacteriuria during pregnancy. With this in mind, the recommended test of cure is a repeat urine culture after completion of the course of appropriate antibiotics, with a repeat course of a different antimicrobial agent if bacteriuria is not cleared. Serial testing

Cystitis with culture is also recommended (by trimester) to detect reoccurrence, with appropriate treatment if the culture is positive. Patients with a persistently positive culture despite treatment should be considered for urologic evaluation and suppressive antibiotics.

Cystitis occurs in approximately 1% to 2% of all pregnancies. Although it rarely leads to pyelonephritis, patients may present with symptoms that are also suggestive of preterm labor.

Diagnosis

The most specific symptom that should lead to clinical suspicion is dysuria. On urinalysis, hematuria and pyuria may be present, although these could also be caused by vaginal contamination of the specimen. A culture with more than 100,000 organisms/mL is the most specific diagnostic sign.

Treatment Similar to asymptomatic bacteriuria.

Prevention A culture for test of cure is recommended, as with asymptomatic bacteriuria, together with repeat surveillance (each trimester) for the remainder of the pregnancy by use of urine cultures. Approximately 25% of patients with cystitis experience another urinary infection during pregnancy. After a second episode of cystitis during pregnancy, suppressive antibiotics are appropriate for the remainder of the pregnancy.

Pyelonephritis

Acute pyelonephritis occurs in 1% to 2% of all pregnancies, with approximately two thirds occurring in patients with previous asymptomatic bacteriuria. The risk for pyelonephritis is significantly increased during pregnancy, probably secondary to physiologic changes in the urinary tract. It is more common in the second and third trimesters. The major significance, in addition to maternal illness, is the increased risk for preterm labor.

Diagnosis

Patients present with systemic signs, including fever, costovertebral angle tenderness, nausea, and vomiting; laboratory evaluation reveals pyuria with leukocyte clumps and bacteria, and elevated leukocyte count. Severe disease can result in sepsis, renal failure, and acute respiratory distress syndrome.

Treatment

Hospitalization is often considered for intravenous (IV) antibiotics; hydration (for dehydration); and close observation for systemic complications, such as preterm labor. After blood and urine cultures are obtained, IV therapy with a broad-spectrum penicillin or cephalosporin is appropriate.

In selected patients, outpatient treatment with two doses of intramuscular (IM) ceftriaxone (1-2 IM every 24 h), followed by 2 weeks of oral antibiotics, has shown similar outcomes to inpatient management. Aminoglycosides may be used with caution during pregnancy.

Sonography should be used to investigate the urinary tract for significant obstruction; if found, urologic consultation is recommended. Spiking fevers are the usual pattern, but patients can be converted to treatment with an appropriate oral antibiotic once afebrile for 24 to 48 hours and continued to complete a 14-day course.

Prevention

Detection and treatment of asymptomatic bacteriuria can prevent as many as two thirds of cases of pyelonephritis in pregnancy. When a patient has completed 14 days of antibiotics therapy, the urine should be cultured again to prove bacteriologic cure. Suppressive antibiotics, such as nitrofurantoin, 100 mg at bedtime, or ampicillin, 500 mg twice a day, should be used for the remainder of the pregnancy[18,19,20,21]

References

1. Lazzarotto T, Spezzacatena P, Pradelli P, Abate DA, Gabrielli L, Varani S, Landini MP. Cytomegalovirus infection in pregnancy. A still complicated diagnostic problem. Intervirology 1998; 41(4-5): 149-57.
2. Nathwani D, Maclean A, Conway S, Carrington D. Varicella infections in pregnancy and the newborn. A review prepared for the UK Advisory Group on Chickenpox on behalf of the British Society for the Study of Infection. Journal of Infection 1998; 36 (1): 59-71.
3. Cox SM, Cunningham FG, Luby J. Management of varicella pneumonia complicating pregnancy. Am J Perinatol 1990; 7: 300.
4. Chapman S, Duff P. Varicella in Pregnancy. Semin Perinatol 1993; 17: 403.
5. Smego RA, Asperilla MO. Use of acyclovir for varicella pneumonia during pregnancy. Obstet Gynecol 1991; 78: 1112.
6. Pastuszak AL, Levy M, Schick B, et al. Outcome after maternal varicella infection in first 20 weeks of pregnancy. N Engl J Med 1994; 330: 901.
7. Centers for Disease Control and Prevention. Prevention of varicella. Recommendations of the Advisory Committee on Immunization Practices Advisory Committee (ACIP). MMWR Morb Mortal Wkly Rep 1996; 45: 1
8. Gilbert GL. Parvovirus B19 infection and its significance in pregnancy. Communicable Diseases Intelligence 2000; 24 Suppl: 69-71.
9. Markenson GR, Yancey MK. Parvovirus B19 infections in pregnancy. Seminars in Perinatology 1998 Aug; 22(4): 309-17
10. Michielsen PP, Van Damme P. Viral hepatitis and pregnancy. Acta Gastroenterologica Belgica. 1999; 62(1): 21-9.
11. Duff P. Hepatitis in Pregnancy. Seminars in Perinatology. 1998; 22(4): 277-83.
12. Munoz FM; Englund JA. Vaccines in pregnancy. Infectious Disease Clinics of North America, 2001; 15(1): 253-71.
13. Isselbacher KJ, Braunwald E, Wilson JD, (eds): Harrison's Principles of Internal Medicine, ed 13. New York, McGraw-Hill, 1991.
14. Eskild A; Oxman A; Magnus P; Bjorndal A; Bakketeig LS. Screening for toxoplasmosis in pregnancy: what is the evidence of reducing a health problem? Journal of Medical Screening. 1996; 3(4): 188-94.
15. Peyron F, Wallon M, Liou C, Garner P. Treatments for toxoplasmosis in pregnancy. Cochrane Database Syst Rev. 2000; (2): CD001684.
16. Vo QT, Stettler W, Crowley K. Pulmonary tuberculosis in pregnancy. Prim Care Update Ob/Gyn 2000; 7: 238.
17. Ormerod P. Tuberculosis in pregnancy and the puerperium. Thorax 2001; 56(6): 494-9.
18. Mikhail MS, Anyaegbunam A. Lower urinary tract dysfunction in pregnancy: a review. Obstet Gynecol Surv 1995;50:675-83
19. Stamm WE, Hooton TM. Management of urinary tract infections in adults. N Engl J Med 1993;329:1328-34.
20. Gratacos E, Torres PJ, Vila J, Alonso PL, Cararach V. Screening and treatment of asymptomatic bacteriuria in pregnancy prevent pyelonephritis. J Infect Dis 1994;169:1390-2.
21. Antimicrobial therapy for obstetric patients. ACOG educational bulletin no. 245. Washington, D.C.: American College of Obstetricians and Gynecologists, March 1998;245:8-10.

TELEMEDICINE - DOCTORS WITHOUT BORDERS

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Introduction

Telemedicine has been defined as the use of telecommunications to provide medical information and services[1]. Telemedicine in its simplest form may involve two doctors discussing a case over the telephone, or may be as sophisticated as using satellite technology to enable a surgeon in one country to remotely control a robotics arm for surgery on a patient in another country (Telesurgery).

The transfer of electronic medical data (i.e. high resolution images, live two-way audio and video, and patient records) from one location to another utilises a variety of telecommunications technology, including ordinary telephone lines (POTS), ISDN, broadband, high-bandwith T-1 lines, the Internet, and satellite link-ups.

Telemedicine involves two different kinds of technology:

The first, called **Store-and-Forward** technology, is used for transferring digital images from one location to another. A digital image is first acquired ('stored') by medical imaging or digital camera equipment and then sent ('forwarded') to another location within the hospital, between two hospitals in the same city, or from one hospital to another anywhere in the world. This technology is typically used for non-emergency situations, when a diagnosis or consultation may be made in the next 24-48 hours and sent back.

Teleradiology, the exchange of X-ray, CT, or MRI images (store-and-forward images) using a Picture Archiving and Communication System (PACS), is the most common application of telemedicine in use today. Teleradiology has enabled a single radiologist to provide consultation to a number of hospitals simultaneously by working out of a central location where images are sent directly for diagnosis.

Other medical specialities such as pathology, where images of slides may be sent from one hospital to another for diagnostic consultation (Telepathology), also lend themselves well to this store-and-forward technology.

Medical transcription[2] may also be considered to be a store-and-forward form of telemedicine.

Face-to-Face technology uses videoconferencing equipment for a 'real-time' consultation between the patient at one location and a specialist at another or a virtual discussion between specialists at different hospitals maybe even in different countries. Typically clinical specialities benefit more from this virtual consultation technology, like Telesurgery which, as noted earlier, has enabled surgeons to remotely control a robotics arm for surgery.

Telemedicine involving a combination of store-and-forward, interactive, audio, still and video images can provide access to specialty care where none was previously available.

History of Telemedicine

The recent explosion of interest in Telemedicine has led to the misconception that it is a relatively new use of telecommunications technology, but the truth is that telemedicine has been in use in some form or other for over thirty years[3].

The Nebraska Psychiatric Institute was one of the first facilities in the USA to have closed-circuit television in 1955. In 1964 a two-way link was established between the institute and Norfolk State Hospital, 112 miles away. The link was used for education, and for consultations between specialists and general practitioners.

The Massachusetts General Hospital/Logan International Airport Medical Station established in 1967 enabled physicians at MGH to provide occupational health services to airport employees and to deliver emergency care and medical attention to travelers using a two-way audiovisual microwave circuit.

The National Aeronautics and Space Administration (NASA) in the United States played an important part in the early development of telemedicine. NASA's efforts in telemedicine began in the early 1960s when physiological parameters were telemetered from both the spacecraft and the space suits during missions. These early efforts and the enhancement in communications satellites fostered the development of telemedicine and many of the medical devices in the delivery of health care today.

The North-West Telemedicine Project was set up in 1984 in Australia to pilot test a government satellite communications network (the Q-Network). The project

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goals were to provide health care to patients in five remote towns south of the Gulf of Carpentaria.

The Memorial University of Newfoundland (MUN has been active in international teleconferencing and in 1985 it became involved in the International Satellite Organization (Intelsat) project linking the School of Medicine at the University of Nairobi with Kampala, which later was extended to six Caribbean countries.

The NASA SpaceBridge to Armenia/Ufa in 1989 conducted the first international telemedicine program for medical consultation between the site of a massive earthquake in the Soviet Republic of Armenia and several medical centers in the U.S.

In India, the Indian Space Research Organisation (ISRO) launched a telemedicine project on 3rd July, 2002 linking the G.B. Pant Hospital in the Andaman & Nicobar Islands with the super specialty Sri Ramachandra Medical College & Research Institute in Tamil Nadu. The Apollo-SHIR project links the Apollo Hospital in Chennai with ISRO's SHAR Hospital in Sriharikota and Aragonda Apollo Hospital in Andhra Pradesh. Similar projects have been undertaken in the Lakshwadeep islands, Assam, Orissa and Tripura[4].

Advantages of Telemedicine

- Telemedicine can make prompt specialised care more accessible to underserved remote populations, especially in developing countries.
- Physicians doing clinical research can be linked together despite geographical separation.
- Videoconferencing also allows continuing education training or professional meetings for isolated or rural health professionals.
- Video consultations from a remote clinic to a specialist can help patients save time and avoid prohibitive travel and associated costs.
- In the context of distant and rural areas, delivery of telemedicine-based medical care is also highly cost effective.

Barriers to Telemedicine

- Most remote areas do not have the high bandwidth telecommunications links required for telemedicine, so ironically those who could most benefit from telemedicine do not have access to it.
- Regular telephone lines do not supply adequate data-communication speeds for real-time telemedicine applications.

Future trends

Innovations in telecommunications technology coupled with the ever expanding applications of telemedicine now allow medical professionals to provide timely and quality services to patients with limited access to health care through utilization of technology.

In the West, competition among technology manufacturers and telecommunications companies have significantly lowered the costs of equipment and bandwidth. With ever-increasing penetration and continuous high-end telecommunications technology advancement, Telemedicine is poised to become a major industry in the health care field.

In Third-world nations low-cost store-and-forward technology can provide medical care to remote areas. It is up to the respective governments to provide the required infrastructure especially in the form of high bandwidth telecommunications access.

In Kashmir, where even accessing the Internet over slow dial-up telephone connections is an ordeal, Telemedicine would seem to be a remote possibility (pun unintended). However, a pioneering basic Teleradiology project using the Internet to discuss Spiral CT and MRI images from the Diagnostic Imaging Centre of SMHS Hospital with radiologists from western countries is currently underway[5].

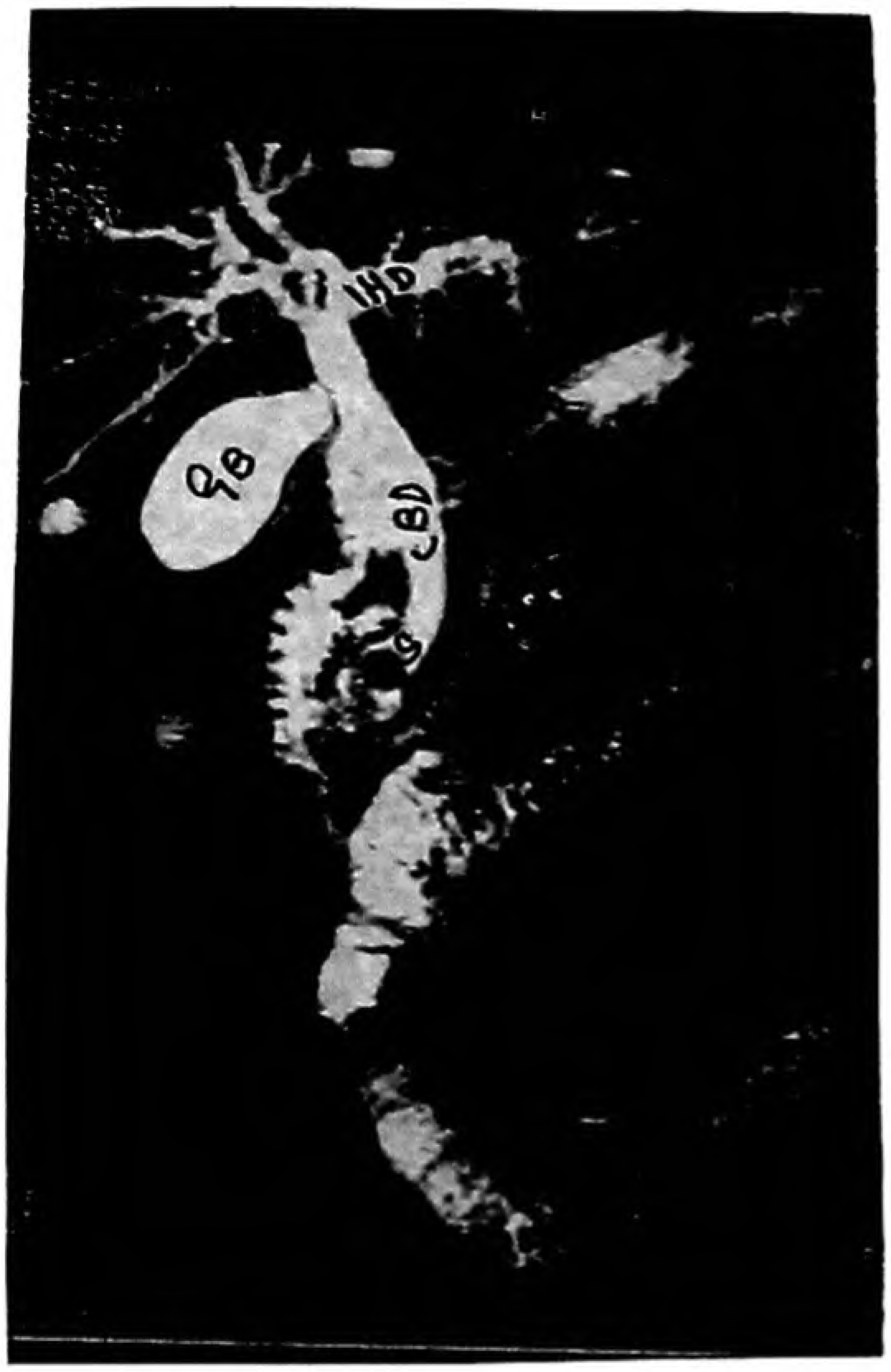
Very recently the Indian Space Research Organisation (ISRO) has undertaken a project to remotely link the Leh District Hospital in Ladakh with the All India Institute of Medical Sciences (AIIMS) via satellite[4].

Telemedicine in Kashmir thus already exists at both the low-tech and cutting-edge levels. With dedicated official support, tackling the the problem of shortage of specialists in remote areas by proper utilization of this technology may hold a great deal of promise.

References

1. Perednia DA and Allen A. Telemedicine technology and clinical applications JAMA Feb 8 [1995] : 273(6): 483-8
2. Jehangir B and Nazir A. Medical transcription : Telemedicine or just teleworking. JK-Practitioner [2001] : 8(2): 119-23
3. A Brief History of Telemedicine http://tie2.telemed.org/telemed101/topics/telemedicine_history.asp
4. Telemedicine in Andaman http://andaman.nic.in/telemedicine.htm
5. SMHS Telemedicine Project (STP) http://www.kashmirtoday.net/smhs/stp.html.

MRCP IN OBSTRUCTIVE JAUNDICE



A young adult presented as Obstructive Jaundice clinical diagnosis of CA head of Pancreas was made. MRCP reveals dilated IHBR and CBD. A calculus is seen at lower end of CBD. The Pancreatic duct is normal.

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VESTIBULAR SYSTEM, BRIEF ANATOMY & CLINICAL IMPLICATIONS

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Introduction

The Vestibular system has been studied in detail since centuries. The system is concerned primarily with balance of the body. The system in collaboration with the cochlear system form system, the vestibulocochlear system. This system is highly developoed in mammals & among mammals still higher developed in the humans. The importance of the vestibular system to the body is not only because of the fact that it is concerned with the balance ofthe body but has effects generalized due to the connections of vestibular system with other well developed parts of the Central Nervous System. this relation with the other parts of CNS makes its significance felt in noting brainstem reflexes, diagnosis & management of coma, in interpratation of clinical signs etc.

Text:

The rule of five applies to the vestibular system i.e.:

- Five receptors
- Five nuclei
- Five tracts
- Five effects

The vestibular nerve brings impulses which are useful in:

- Coordinating Reflexes of Eyes
- Coordinating Reflexes of Neck
- Coordinating Reflexes of Body

Receptors:

The five receptors are:

1. Crista ampulla of superior Semicircular canal
2. Crista ampulla of lateral Semicircular canal
3. Crista ampulla of posterior Semicircular canal
4. Macula of utricle
5. Macula of saccule.

These receptors (hair cells) in these structures reach vestibular ganglion in the internal ear.

The axons of vestibular ganglion form vestibular nerve which passes through internal auditory canal to reach pontomedullary junction.

The vestibular nerve ends at five places:

1. Medial Vestibular nucleus.
2. Lateral Vestibular nucleus
3. Superior Vestibular necleus
4. Inferior Vestibular nucleus
5. Direct fibres to cerebellum.

From these vestibular nuclei, five tracts arise going to different parts of CNS.

1. Vestibulo ocular tract.
2. Lateral vestibulospinal tract
3. Medial vestibulospinal tract
4. Indirect vestibulocerebellar tract.
5. Vestibuloreticular tract.

Vestibulo ocular tract.

this tract goes to end in the nuclei of 3,4 & 6th Cranial nerves. These three cranial nerves move the eye.

The reflex set up in the vestibulooccular reflex helps to make the eye able to remain firxed on stationary objects while the head end of the body is moving. When the head is turned slightly to the right eyes are turned to the left the exact distance so that the field of vision remains the same.

Stimulation of the vestibular nuclei on the right side causes conjugate deviation of the eyes to the left. The assessment of vestibuloocular reflex is used in the assessment of patients in Coma. With the help of Dolls head manouvre or the ice water caloric test.

In coma resulting from any cause, the eyes move easily from side to side in a direction opposite to the direction of head turning. the ease with which the eyes move towards the opposite side is a reflection of the disinhibition of the brainstem reflexes by the damaged cerebral hemispheres in the assessment of depth of coma.

Secondly the conjugate ocular movements demonstrate the integrity of the brain stem pathways.

thus full & conjugate movements of the eyes that are induced by the occulocephalic manouvre demonstrate the intactness of a large part of brain stem & exclude a primary lesion in the brain stem as a cause of coma, suggesting a lesion elsewhere.

Caloric stimulation of Vestibular apparatus also acts as an adjunct to the above reflex. Irrigation of the external auditory canal with cool water stimulates the endolymph of the labrynths of inner ear.

An intact brainstem pathway is indicated by tonic deviation of both eyes towards the side of cooling water

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irrigation. Bilateral conjugate movement therefore have same significance as occulocephalic reflex.

Vestibulospinal tracts: Lateral & Medial)

The impulses descending in these tracts strengthen the extensor muscles of the limbs providing enough power to support the body against gravity & maintain an upright posture. Interruption of the brainstem leads to a condition in which the legs are held in stiff position of extension referred to as decerebrate rigidity.

Cutting the brain stem or damage to the brain stem at midbrain stops inhibitory tracts which descend from above this level & allows vestibular reflexes to send continuous impulses to ant. Horn cells of sp-inal cord through vestibulospinal tracts which normally act to strengthen the Extensors. This happens in azotemia, anoxia, drug ingestions, damage to corticospinal tracts or damage to caudal diencephalon.

Vestibulocerebellar tract:

this tract from the vestibular nucleus ends in the part of cerebellum called the flocculonodular lobe of Cerebellum. Damage to this tract or to the Flocculonodular Lobe leads to imbalance & a staggering type of gait with a broad base. These persons cant walk normally & tend to fall frequently & require a support to walk. It is primarily concerned with the balance of the body.

Vestibulo Recticular tract:

This tract ends in the Reticular formation area & more specifically in the area prostema, a v shaped area on the lateral wall of the fourth ventricle. The function of this tract evident in people suffering from motion sickness or sea sickness.

Stimulation of the vestibular apparatus because of excessive motion causes motion sickness. This comprises of nausea, vomiting & vertigo induced by irregular or rythmic movements.

Thus we have seen the importance thast the Vestibular System has in the body. A single system having deep rooted connections & manifesting in the body ailments by various clinical signs. A way of seeing the mastery of the Almighty.

NEED FOR HEALTH CARE REFORMS

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The universal goal of the healthcare system is to ensure adequate access to quality care at a reasonable price. The components of this goal include access, quality and price. Each of these factors cannot be dealt with in isolation. Each component would be maximized with the goal of improving health. The purpose of health services is to secure through the resources available the greatest possible improvement in the physical and mental health of the people. This warrants health services to bring healthcare closer to where people live, become more responsive to the needs and wishes of patients, get the most from the resources committed to it.

We all need to take stock of what humankind has achieved during the past two millennia. We also need to assess the relative significance of the achievements of the past fifty years compared with the previous 2000 and our expectations for the next 20 years. The practice of medicine is becoming increasingly complex. It combines both science and art. Science-based technology is the foundation for the solution to many clinical problems. The combination of medical knowledge, intuition, and judgment defines the art of medicine.

No healthcare system in the world is stable. All systems would undergo considerable change in next twenty years. Change is being driven by the growth of the middle class, greater demands from that middle class, and the globalization of economies.

Health services comprise of medical care and public health services and are a function of the political system of a community. Political forces/ideologies play a major role in determining the health policies of the country through decisions on resource allocation, human resource policy, choice of technology and the degree to which health services are to be made available and accessible to the population. Health policies can also be used as a political lever to promote certain vested market invests.

The hospital is the predominant institutional provider. The hospital consumes the largest single share of personal healthcare resources and in some respects is the most visible and central organization in modern healthcare. Recently, the growth of the ambulatory care, especially within the context of technological advances and cost pressures, is causing the hospital to lose its historical pre-eminence. It has to evolve from the centre of excellence to community support.

Humanity has undoubtedly achieved a great deal over the last 2000 years. Hospitals have benefited from the new technology, but have increasingly found them selves unable to keep up with the pace of the technology change. Today, hospitals are competing to acquire cutting-edge technology that, because of the relentless pursuit of technological perfection, will be obsolete within a few years. Perfection becomes a utopian dream that is simultaneously too expensive to achieve and expensive not to pursue. The only means of achieving this dream is to ensure we have the latest technology at all times. Healthcare needs and medicine's capacity to respond are not static. It is necessary to determine the true current state of hospitals in order to increase their capability to respond to the population's needs. A key element is to develop and test a methodology for describing and comparing the state of hospital systems. It will begin to identify common issues and opportunities, with important policy implications.

Ambulatory care includes a wide range of services for the "walking' or non-institutionalized patient. Ambulatory care services play a key role as the initial and continuing point of contact with the system for more people. . It also serves as a centre of the referral network for specialized hospital care. . An increasing number of people have sought primary care from hospitals, sometimes because of lack of access to other care, taxing the ability of many facilities to respond. The result has been overcrowded facilities; the wrong mix of services, equipment, and personnel to respond to patient needs; and extremely dissatisfied consumers and providers. A hospital-based ambulatory care can provide comprehensive and accessible care and remove primary care patients from emergency rooms. Many hospitals have now successfully expanded outpatient services.

Long-term or chronic care, along with mental health services, represents one of the greatest challenges for the future in healthcare. Mental health services involve a number of unique issues. Our society has yet to accept responsibility for

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solving the problems involving the mental health of our nation's citizens.

The emphasis on both practice guidelines and evidence-based practice has yielded a further orientation towards outcome measures as indicators of community health. Accurate, comprehensive, and timely community health data are essential requisite of goal setting and programme performance monitoring. Health information is vital. The information revolution should allow better programme management and assessment. Information technology and consumerism will transform healthcare worldwide.

A system approach to healthcare is used to emphasize the interdependencies among the parts of the system and between the system's 'hard assets' and the organizational and financial arrangements that allow the system to function. A solid understanding of the system is essential for policymaker, provider and consumer. The dynamic and interactive nature of the system should not be forgotten during planning and policymaking. Efforts to improve services should be based on modifying each element in the system. Today's health policy decision-makers must focus on health behaviour and health behavioural change to address contemporary health issues. The changing nature of the external environment, patterns of illness, and of the system's necessary responses should be outlined.

Health is important among the objectives and values of most individual human beings and that they expect their government and administrations to pursue policies that will afford them the opportunity to attain lives of optimum duration and quality. The health of its citizens is one of the most important resources needed by a nation for the pursuit of most other legitimate national objectives. National human behaviour requires some commensuration of the benefits and costs of all the options we seek to choose. India has experienced a faster growth in demand for health and medical care than in the resources it can easily make available for meeting that demand. Demographic and epidemiological changes, technological advances, the emergence of new threats to public health and the rising aspirations for a better life that are part of the evolving political development of our species, all sharpen the need to make choices and decisions about how the responsibilities and rewards of human activity are to be distributed. The public health is important enough to warrant planning for it.

Improvement in health and reduction in death rates have occurred at most stages in life from birth through to old age. We live, on average, nearly as long as our counterparts in most other middle economies and developing nations. There is need to identify those public health priorities where there is sufficient evidence to conclude that action would have a significant benefit on health. Strategies for action must follow. Priorities must include those where both the evidence and the support for action is strong, those where the case for action is strong, but more evidence of the feasibility of implementation is still needed, and those where the case for action is strong, but where detailed recommendations are yet to be evolved.

The health of the people of India is now almost better than it has been prior to independence. Despite the progress made during the last five decades, grey areas still exist - inequality in health, social class and disease, quality of care, ever-increasing costs, etc. However, complacency cannot be justified. The aim of Healthcare delivery system and health policy should be to embrace the health of the public and protect and promote the health of all citizens in the interests both of those individuals and of the community, they comprise. It involves the promotion of health, the prevention of disease, the treatment of illness, the care of those who are disabled and the continuous development of the technical and social means for the pursuit of these objectives.

The main objective of National Health Policy 2002 is to achieve an acceptable standard of good health amongst the general population of India. The approach would be to increase access to the decentralized public health system by establishing new infrastructure in deficient areas, and by upgrading the infrastructure in the existing institutions. Emphasis would be given to ensuring a more equitable access to health services across the social and geographical expanse of the country. Attention will be paid to increasing the aggregate public health investment through a substantially increased contribution by the central government.

The health needs of the country are enormous and the financial resources and managerial capacity available to meet them fall somewhat short. National Health Policy had to make some hard choices between various priorities and operation options. The policy, while being holistic, undertakes the necessary risk of recommending differing emphasis on different policy components. The need to ensure that "equity" in the health sector stands as an independent goal has got desired attention.

An expectation of significant improvement in the quality of services, and the consequential improved health status of the communities depends not only on increased financial and material outputs but also on a more empathetic and committed attitude in the service providers.

The health services would be expected to be responsive to the needs of all; cost-effective and affordable, and of high quality; humane, caring, and culturally acceptable; innovative in provision of services and exploitation of technology; sensitive to the wide spectrum of determinants of health (from nuclear biology to socio-economics) and the environment; based on sound ethical principles that define individual responsibility as much as the state's obligation, and able to measure the outcome of the investments.

The WHO has identified four priority areas for future action: ensuring value for money, poverty reduction, public health policy and strengthening national capabilities for emergency relief and humanitarian assistance.
There is universal trend towards greater decentralization, pro-market economic policies, rapid expansion of the private health sector, introduction of user charges, and purchaser-provider splits. Developing countries like India are still struggling to expand their health infrastructure and provide basic health facilities to the entire population. If India is to progress towards equity, equality, comprehensiveness, universal accessibility and coverage, radical institutional reforms in the health sector are needed. The potential advantage from restructuring health care is that it can be extended to those who need it most.

The current era has been a time of clear change for health care delivery systems. Recent experiences of developed countries suggest that investment in decentralized population-based systems and health services research aimed at the developing evidence-based healthcare, rather than the emphasis on central, disease-based systems could yield substantial health gains for the populations. Despite some progress made in reduction of birth rate, and mortality rate, and increased life expectancy, India continues to have a poor performing health system as compared to Sri Lanka, China or Singapore. Primary care is not yet available to every citizen because of poor planning and implementation. Even after 55 years of independence India does not have a National Health Service and is nowhere close to achieving Health For All for its people. About 40% of population lives below poverty line and an equal number of people do not have access to even basic health care. We should learn from the tigers taking every opportunity to improve healthcare.

Emphasis should be directed on developing an enabling policy and institutional environment in the health sector, and promoting an effective health dimension to social, economic, environmental and development policy. The objective should be to reduce child mortality; improve maternal health, combat HIV/AIDS, malaria, tuberculosis and other diseases, and provide access to affordable, essential drugs. More attention should be paid towards sustaining development and reducing poverty. A healthy population is a prerequisite for growth as much as a result of it. Investing in the health of poor people and promoting pro-poor health policies and practices should be a top priority. Precious investments in people's development lead to health equity and well-being.

Education and Health is the foundation for building a strong republic. The development of health is a holistic process related to the overall growth and development of social, cultural, economic, educational, political and environmental factors. Health also depends on many supportive services, such as water supply, sanitation, nutrition, public health and medical services, and human resources. Health policies are influenced by the policies concerned with these areas. For instance, development policies to eliminate poverty and inequality are directly linked to the health policy, population policy, education policy and other social development policies.

It needs strong political will and imagination. Although organizing quality care for all citizens is a responsibility of the government, the public has important responsibilities. Health is everybody's right and everybody's responsibility.
Today's challenging healthcare environment demands that hospitals and health systems continuously monitor, improve and refine how they deliver patient care efficiently. There are problems with the health care system. Many of these problems will require action by policymakers. National health policy has been developed for many years on a fragmented, incremental basis. Our nation must choose wisely to ensure both high quality care and access for all of our citizens. Prevention and health is everybody's right and responsibility.

Further Readings:

1. Amin Tabish. Textbook of Hospital & Health Services Administration. Oxford University Press. New Delhi. 2002.
2. Amin Tabish. Looking after Health into the 21st Century. JK Practitioner. 1998; 5(2): 156-158.
3. Amin Tabish. Maternal & Child Health in developing countries: responding to the challenge. J Applied Med. 1998; 24(8): 579-584.
4. Amin Tabish. Health Services Organization & Public Health Delivery in India – Part I. J Applied Med. 1999; 25 (4): 243-248.
5. Amin Tabish. Health Services Organization & Public Health Delivery in India – Part II. J Applied Med. Sci. 1999 25(5): 323-326.
6. Amin Tabish. Towards Development of Professional Management. J Management in Medicine, England. 1998; 2(2): 178-187.
7. Amin Tabish. Holistic Approach to HFA By 2000. World Health Forum. Geneva. 1993; 14(2): 173-174.
8. Amin Tabish. Evidence-Based Medicine: linking research to practice. J Med Sci. 1999; 2(1): 2-3.
9. Amin Tabish. The Electronic Highway in Medicine. JK Practitioner. Editorial. 1999 6(4): 251.
10. Amin Tabish. Tackling Emerging Infectious Diseases. J Med Sci. 1999; 2(2): 12-16.
11. Maxcy-Rosenau-Last. Public Health & Preventive Medicine - 14th. ed. . Prentice-Hall International Inc. USA 1998.
12. Richard Smith. Editorial. The Future of healthcare systems. BMJ. 314 24 May 1997.

1. 4th. NATIONAL CONFERENCE OF THE SOCIETY OF GI ENDOSCOPY OF INDIA (SGEI)

A two day conference "ENDOCON 2003" organised by SGEI was held at scope convention complex, New Delhi.One day was reserved for live demonstration of endoscopic practices on GI (pancreatic and biliary) maligancies. Eminent experts like Paul Swain (UK),T Ponchum (France), S Bhargava (USA) attended the conference. Doctors from Bombay, Hyderabad Apollo and Ganga Ram Hospitals namely Dr.DN Reddy, Dr. Ravinder Saud, Dr.Ajay Kumar, Dr.Amit Maydeo gave live demonstration on GI Therapeutic procedures.

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Besides lectures, panel discussions were chaired and moderated by Prof. Kocher, Prof Basin, Prof. RK Tondon, Prof Bhargava, Prof. Sharma, Prof.MS Khuroo, Prof. Zargar, Dr. Vipul Rather and others.

Dr. Ajay Kumar & Dr.Ravinder Saud (Course Directors) put all efforts to make this conference a successful event.All arrangements were excellent.The delagates enjoyed the academic event especially the lecture by Dr.Paul Swain, the inventor of capsule endoscopy. A full day (Day 2 nd) was devoted to GI bleeding. Prof. Khuroo, Prof. GM Malik, Prof. Zargar, Dr.BA Khan & Dr.Gulzar were invited delagates from J&K State.

Prof.GM Malik MD, FACG
Delegate.

2. NC-ASICON-2003

The 23rd. Annual conference of the nothern Chapter of Association of Surgeons of India was organised by Mohan Oswal Cancer Treatment and research Foundation of Ludhiana from 13th-16th March 2003. A team of surgeons from the Kashmir Valley participated in the conference.

Prof.Muneer Khan had the honour of chairing a session. It was for the first time that a good number of scientific papers were presented at ASICON by the valley surgeons. The presentations were highly appereciated by the gathering. Two teams of PG students comprising of Dr.Shiekh Tariq Ahmad\Dr.Arshad Hussain and Dr.Ajaz ahmad rather/Dr.Rafiq Simnani made it to the final round of the quiz contest. Dr.Nissar Ahmad Wani was unanimously elected as an executive member of NC-ASICON.

Due to the efforts of Prof. Muneer Khan,Dr. Nazir Ahmad Salroo,Dr.Nissar Ahmad Wani and Dr.Iqbal Saleem Mir the general body meeting accepted the offer of Govt.Medical college Srinagar to host NC-ASICON-2004 Srinagar in the month of April with Prof.Muneer Khan as organising secretary.

CONFERENCE CALENDAR

1.Indian Society of Gastroenterology,Midterm Conference 2003.
Bangalore ,10-11 May 2003,Hotel Taj West end.
For details:-
Dr.Naresh Bhat,Organising Secretary Department of GIT,St.John's medical college Bangalore-560034.e mail 15gban2003&hotmail.com

2.Annual conference of the Association of Colorectal surgeons of India. 7th Advanced instructional course 2-4 september 2003.National conference 5-7 september 2003 at Ranchi (Jharkhand)
For Details:-
Dr.Satish Midha Organising secretary
Tel. 0651-2282929 Mobile 098351-50204
E mail midhasatish @hotmail.com

3.National conference on Pulmonary disease (NAPCON-2003) 12-16 November 2003.
Venue:- Hotel the Residency and Hotel Rathna Regent,Ciombatore.
For details:-
Dr.T Mohan Kumar,Organising Secretary.
Website:- www.napcon2003.com

CONDOLENCES

The Editor and the editorial board of JK-Practitioner condoles the death of:-

1.Dr.Yamen Ashraf (Consultant Radiology) GMC, Srinagar who died of brain tumour at a young age in february 2003.

2.We also pay condolences to the family of Prof.GR Bacha (MD) founder of MET (J&K State) Sopore and an eminent scholar and social leader who died on 16—3-03 of a massive heart atttack after delivering a speech at Sopore.

May God give peace to their souls.
Prof.GM Malik MD

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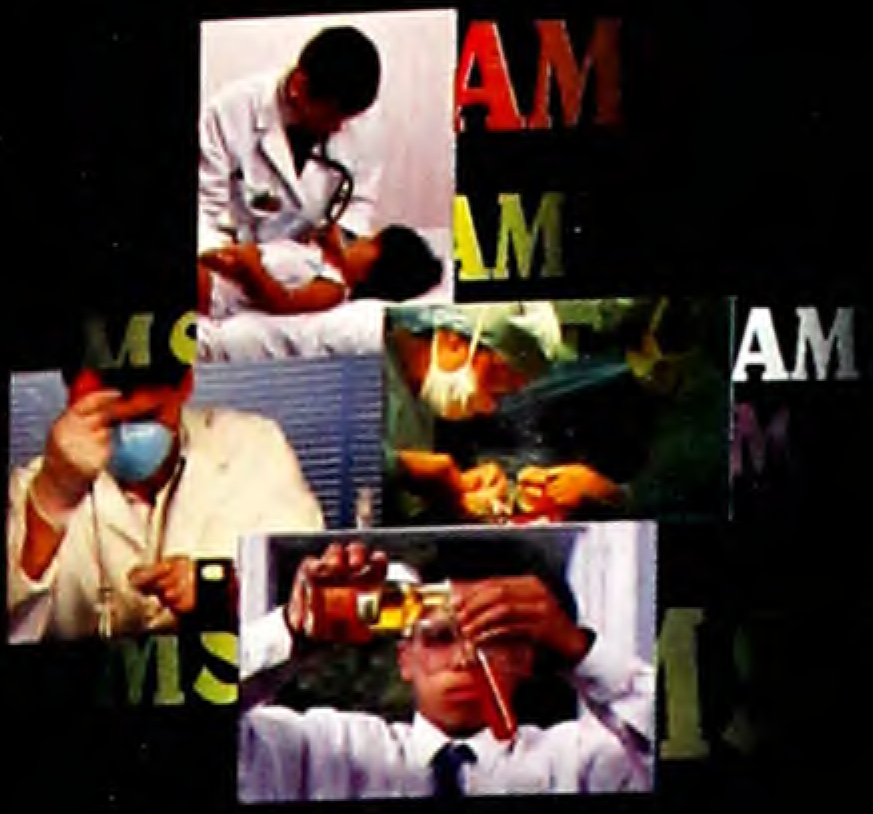

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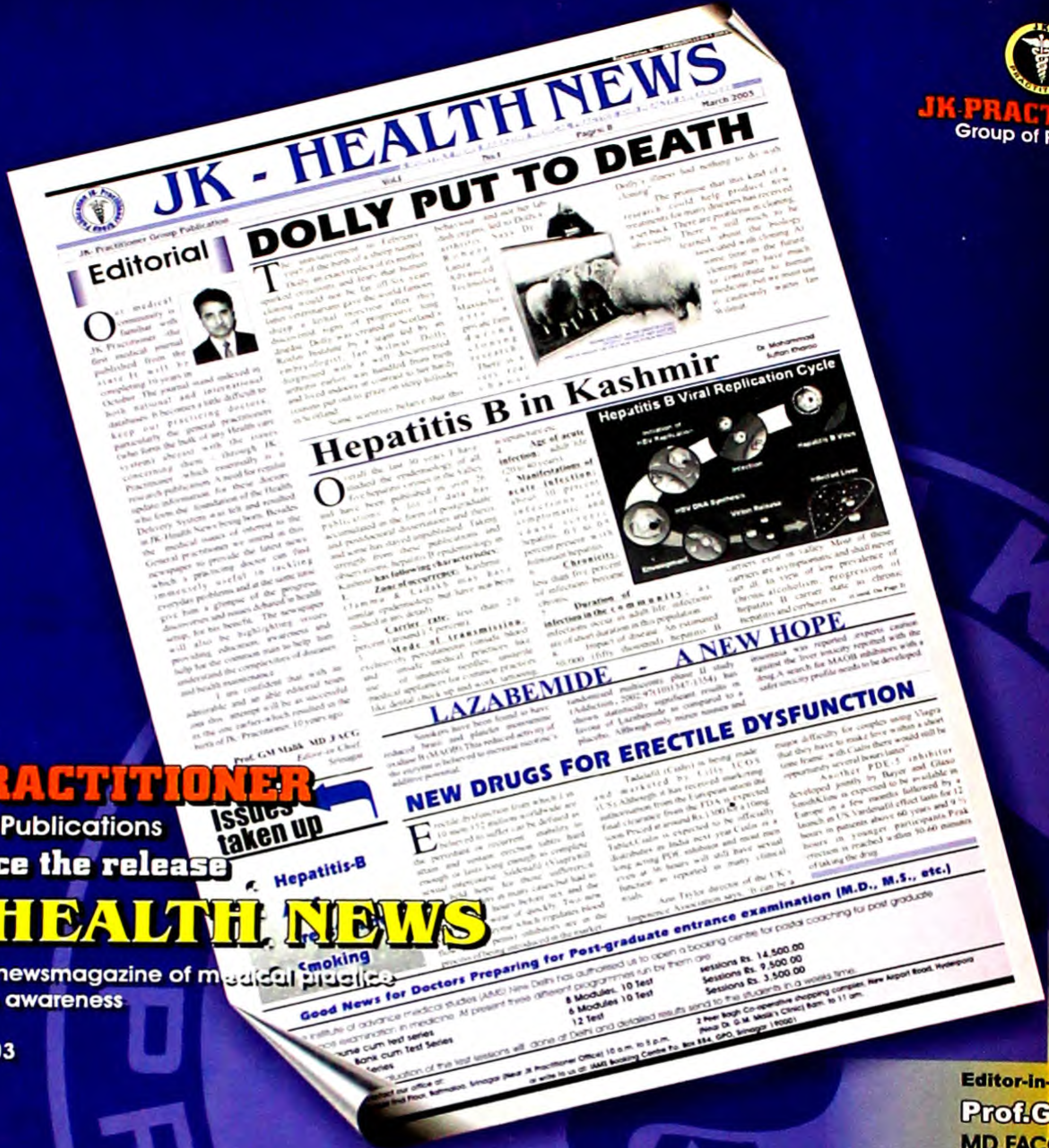

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Our medical community is familiar with , JK- Practitioner the first medical journal published from the state. It
completing 10 years in October. The journal stands indexed in both national and international databases. It bec
little difficult to keep our practicing doctors, particularly the general practitioners (who form the bulk of any Hea
system) abreast with issues concerning them- through JK- Practitioner which essentially is a research publication .A n
these doctors who form the foundation of the Health Delivery system was felt and resulted in JK- Health News bein
Besides the medical issues of interest to the General practitioner we intend in this newspaper to provide the latest news
a practicing doctor can find immensely useful in tackling everyday problems and at the same time give him a glimpse
progress, discoveries and issues debated in health setup, for his benefit. The newspaper will also be highlighting
providing education, awareness and help for the common man to help him understand the complexities of diseases and
maintenance.

I am confident that with an admirable and an able editorial team , our this attempt will be as successful as the one earlier
resulted in the birth of JK- Practitioner, 10 years ago.

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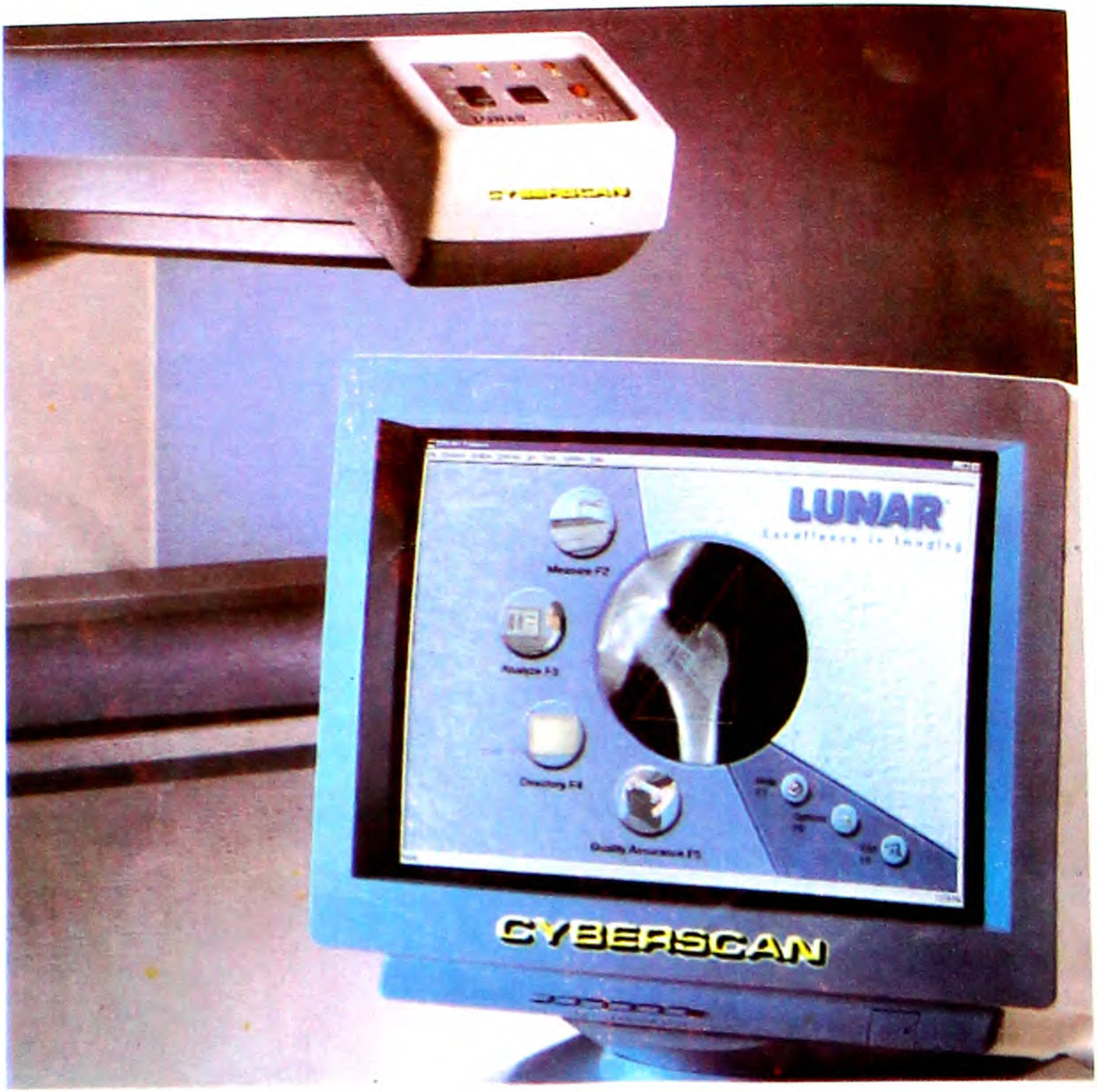
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ANTI PROLIFERATIVE AGENT ELUTING CORONARY STENTS

Since the introduction of angioplasty restenosis has been a major factor limiting the long term success of percutaneous coronary revascularization[1]. The growing use of stents has improved the results of percutaneous coronary revascularization[2]. However, in-stent restenosis continues to limit the long term success of this approach.

The refinement of stenting techniques in the past decade has substantially improved the overall results of the procedure[3]. Despite considerable efforts to prevent the development of restenosis, however, including systemic or local delivery of biochemical substances and drugs[3] and the use of various devices[4] additional target – vessel revascularization is required in more than 15 % of patients. Although catheter based brachytherapy is effective in the treatment of in – stent restenosis[5], its value in the treatment of primary lesions is less clear. Also the use of brachytherapy is limited by its high cost and burdensome instrumentation and the risks inherent in the use of radioisotopes.

Because of these limitations with the above modalities active research was going On targeting the basic pathogenic factors involved in restenosis, i.e. growth factor induced cell proliferation of fibroblasts, vascular smooth muscle cells, etc; which resulted in discovery of anti – proliferating agents like Sirolimus and Paclitaxel. Sirolimus also called Rapamycin (after Rapa Nui the name given to Easter Island by its inhabitants; in the south pacific, a remote, enigmatic place from the culture of whose soil an Actinomycete, Streptomyces hygroscopicus was found to produce by natural fermentation a novel macrolide with potent antifungal ,immunosuppressive & antimitotic activities) is a macrolide antifungal agent with unique anti – proliferative mode of action and powerful immunosuppressant properties . inhibits several regulators of cell – cycle progression and the migration of smooth muscle cells. In 1999, it was approved as an anti – rejection drug in organ transplants. Because of its powerful anti – proliferative, anti - migratory and anti – inflammatory properties, it has been found useful in preventing acute rejection of renal allografts as well as in – stent restenosis.

In a seemingly unrelated development, while Sirolimus was being investigated as an anti – rejection drug, coronary artery stenting was beginning to be used as a means of preventing restenosis after coronary angioplasty. But still, coronary stenting has limitations. Although the procedure was developed to prevent restenosis after angioplasty, it has been an imperfect solution, as 15 – 20 % of stented arteries again become stenotic. Restenosis results from neointimal proliferation in which smooth muscle cells are stimulated to proliferate by inflammatory mediators released in response to vessel injury. Ingrowth of tissue through the struts of the stent eventually narrows the lumen of the stent. Despite numerous attempts to block neointimal proliferation as listed above, no interventions have been found to be consistently successful until now.

On the supposition that immunosuppressive and anti – mitotic properties of Sirolimus might inhibit neointimal proliferation, a stent coated with Sirolimus was devised. This was done by coating the stent with a mixture of synthetic polymers blended with Sirolimus, then applying a second coat of drug – free polymers to serve as a diffusion barrier. The polymer acts as a drug reservoir and allows for the gradual elution of Sirolimus over a period of about 30 days delivering the drug precisely to the site of interest. Only a small quantity of the drug is required and systemic side effects are avoided.

Recently a randomized, double blind trial of comparison of a Sirolimus –eluting – Stent with a standard stent for coronary revascularization (RAVEL study) has been completed with exciting results. In this study at six months follow–up, the degree of neointimal proliferation, manifested as the mean late luminal loss, was significantly lower in the sirolimus stent group than in standard stent group. 0 % of the patients in the sirolimus stant group as compared with 26.6 % of those in the standard stent group had stenosis of 50 % or more of the luminal diameter[5].

Paclitaxel is an anti – neoplastic agent used in various malignancies. Similar studies coated with this agent are in progress and preliminary results are encouraging. The results of the RAVEL (Randomized Study with Sirolimus coated BX Velocity Balloon – Expandable Stent in the treatment of patients with de Novo Native Coronary Artery lesion Study) have already drawn the interest of interventional Cardiologists, but a great deal of work remains to be done. To be widely applicable, these stents will need to be used in complex coronary lesions (as against RAVEL study in which single, primary type A & B lesions in native coronary arteries are selected) and also longer follow – up is necessary to ensure that the excellent initial are durable and the late complications such as stent thrombosis or restenosis at the edges of the stent do not occur. If these points are successfully done with the initial promise of Sirolimus – eluting stents is fulfilled, a new chapter will be opened in the story of this anti – proliferative agent.

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References:

1. Holmes DR Jr, Vliestra RE, et al. Restenosis after PTCA: A report from the PTCA registry of the national heart , Lung, and Blood Institute. Am JCardiol 1984; 53: Supp. 77c – 81c.
2. Sigwart U, Puel J, Mirkovitch V, et al. Intravascular stents to prevent occlusion and restenosis after transluminal angioplasty. NEJM 1987: 316;701 – 706.
3. Serruys PW, VanHout B, et al. Randomized comparison of implantation of Heparin – coated stents with balloon angioplasty in selected patients with coronary artery disease (Benestent II). Lancet 1998; 352: 673 – 81.
4. Kuntz RE, Safian RD, et al. Novel approach to the analysis of re-stenosis after the use of three new coronary devices. J Am Coll Cardiol 1992; 19: 1493 –1499.
5. Teirstein PS, Massullo V, et al. Catheter based radiotherapy to inhibit re –stenosis after coronary stenting. NEJM 1997; 336: 1697 – 703.
6. Marie – Claude Morice, Patrick W. Serruys, et al. Randomized comparison of a sirolimus – Eluting Stent with a Standard Stent for coronary revascularization. (The RAVEL study) NEJM, 2002; 346: 1773 – 1780.

MANAGEMENT OF SEQUELAE OF SEPTIC ARTHRITIS OF HIP IN INFANCY

S.C. Goel M.S., FAMS; Vivek Logani M.S.

HISTORICAL ASPECT

The first well-documented description of septic arthritis in children was given by Thomas Smith, an English surgeon, in 1874[1]. He reported 21 cases of pyogenic arthritis involving primarily the hip joint in infants, 13 of which died and 8 recovered but were crippled. Thus, for many years, this disease was known as Tom Smith's disease. The destructive effect of staphylococcal infection on articular cartilage was first shown by Dallas Phemister in 1924[2]. The contribution of Keefer[3], Curtiss[4], and others in understanding the pathophysiology of septic arthritis cannot be overemphasized. Pressure changes induced in the joint from infection were demonstrated later[5]. Recently, lysosomes as a possible mechanism involved in the pathogenesis of septic arthritis have formed an area of great interest[6,7].

FACTORS INFLUENCING THE OUTCOME OF SEPTIC ARTHRITIS OF HIP

Sequelae of septic arthritis can present in different ways. There are various factors that influence its outcome and presentation.

INTRINSIC FACTORS

Age at onset of Infection:

Most published reports suggest that neonates and infants are more likely to have poor prognosis than older children[8,9]. Premature births are even more likely to fare badly[10,11].

Immunity:

The relative immaturity of immune system in premature births, neonates and young infants makes this group most vulnerable[12]. Abnormalities of immunoglobulins, complements, or phagocytes are present in more than half patients of septic arthritis[13].

Vascular anatomy of femoral head:

Transphyseal spread of infection to the epiphysis through vascular channels occurs in infants up to two years[14,15]. Such vessels gradually get obliterated in older children. Thrombosis of these vessels leads to ischemia and subsequent infective lysis of the growth plate. This, if occurs before ossification of femoral head, may result in complete necrosis and resorption of femoral head and neck ending up in a knob of bone at proximal femur[12].

Intracapsular pressure:

Elevated intracapsular pressure due to infection[5] may cause occlusion and thrombosis of retinacular vessels. It may also predispose to subluxation or dislocation which further compromises the vascular supply of capital femoral epiphysis.

Concomitant Osteomyelitis of Proximal Femur:

This results in a far worse prognosis as compared to infection localized to the synovium of hip joint[11,12,16-19].

EXTRINSIC FACTORS

Infecting Organism:

Most reports have suggested that the most common organism causing septic arthritis is *Staphylococcus aureus* and is usually associated with a worse prognosis than with other infective organisms[8,10-12,16,20-26]. Direct destructive effect of staphylokinase on articular cartilage has also been demonstrated[27-29].

Delay in diagnosis and administering treatment:

It is the single most important factor determining the outcome[10-12,16,17,25,30-32]. Critical period beyond which delay becomes significant has been reported to lie between 4-7 days in various series[10,11,16,19,24].

Adequacy of treatment:

This includes duration and type of antibiotic coverage, post-operative immobilization, drainage and decompression, physiotherapy programme, and follow-up.

SEQUELAE

Even with early recognition and emergency arthrotomy for adequate decompression, the prognosis of septic hip is

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still unpredictable[8,33]. The sequelae of septic arthritis of the hip in infant are diverse and include premature closure of triradiate cartilage, acetabular dysplasia, limb length discrepancy, premature or asymmetrical fusion of proximal femoral physis, subluxation, dislocation, necrosis of the cartilage, ischemic necrosis of femoral head, pseudoarthrosis of the femoral neck, and complete destruction of femoral head and neck[20,21,34-36]. A useful radiographic classification of such sequelae has been given by Hunka et al[35] and later modified by Choi et al[10] as follows (Fig.1):

Type I:

In these hips, the growth of proximal femoral ossification center results in an almost normal hip (Type I A) or in mild coxa magna (Type I B) Delay of ossification, mottling or fragmentation of proximal femoral ossific nucleus is followed by a relatively rapid and complete ossification. The proximal femoral physis remains viable, with little or no shortening of the femoral neck. Acetabular development is adequate, the long term results are good and no reconstructive operations are needed.

Type II:

The physis and metaphysis are involved, with resulting coxa vara or coxa valga (Type II B). These hips usually have radiographic evidence of delay in ossification, flattening and irregularity of femoral head, and coxa magna. The femoral neck is short and wide, and there is relative overgrowth of greater trochanter because of premature closure of capital physis. When there is early symmetrical closure of proximal femoral physis (Type II A), coxa breva with overriding of the trochanter develops, and there is a resultant limp and considerable limb length discrepancy. When premature physeal closure is asymmetrical and incomplete, the femoral neck progressively goes into varus or valgus alignment with secondary acetabular dysplasia.

Type III:

In these hips, the deformity is secondary to injury of the femoral neck, resulting either in angular deformity with severe anteversion or retroversion (Type III A) or in pseudoarthrosis of the femoral neck with complete epiphyseal slipping. (Type III B). These changes result in alteration of acetabular development as well as limb length discrepancy and relative trochanteric overgrowth.

Type IV:

In the most severe cases, compromise of femoral head and neck results either in an unstable hip with a persistent remnant of the femoral neck (Type IV A) or in complete loss of femoral head and neck with no vestige of an articulation of the hip (Type IV B). These hips have the most unpredictable radiographic findings at follow up, with severe limb length discrepancy, acetabular dysplasia, premature closure of triradiate cartilage[29] and marked proximal migration of femur with or without formation of a false acetabulum.

MANAGEMENT

The effective management of sequelae of septic hips of infancy still remains a challenging goal for the orthopaedic community. Nevertheless, many surgical and non-surgical procedures have been described which can improve at least the short term functional status till the patients attain skeletal maturity so that appropriate definitive procedure can be done in an easier and more effective manner.

The components and principles of management are:

- Concentric reduction of hip subluxation / dislocation.
- Preservation of epiphyseal cartilage remnants[37,38] and reorientation to the axis of normal physiological load.
- Correction of deformity.
- Achieving stability of hip joint.
- Adequate containment of femoral head.
- Correction of limb length discrepancy.

Conservative treatment

The main indications for conservative treatment[10,21,35,37] are:

- Mild coxa magna without angular deformity (Choi Type I B).
- Radiological features of resolving avascular necrosis of femoral head which has not yet undergone major deformation (Choi Type I B).
- Coxa breva without angular deformity (Choi Type II A).

The various modalities include abduction cast, bracing etc. for an adequate period of time till reossification is considered sufficiently advanced for unprotected weight bearing[10]. Usually, such methods are successful if applied as early as possible (especially within 2 months of septic process) and when head is still well formed[21]. Regular follow up is essential[10,21] to detect persistent instability, relative trochanteric overgrowth, acetabular dysplasia, subluxation or dislocation, limb length discrepancy etc. which must be treated accordingly (see below). Recently, femoral neck lengthening osteotomy after growth disturbance of proximal femur has been described[39].

Surgical treatment

The various surgical problems related to sequelae of septic hips and available modalities of treatment are outlined below:

Progressive Coxa vaxa with limb length discrepancy

(Choi Type II B)

Most common type of instability of hip is coxa vara, since inferomedial aspect of femoral head and neck is usually spared[21]. In such a case, valgus osteotomy of proximal femur can restore normal joint mechanism both for physiological strain pattern and effective abductor movement[10,35,37]. It also compensates for some of the limb length discrepancy depending upon neck length and angle of correction[40]. Extension osteotomy may also be done concurrently to correct flexion deformity of hip (if any). After correction, remodeling of the hip joint to a better congruence develops to a certain extent, although remodeling potential of the hip with septic sequelae is less than that in developmental dysplasias[29]. Trochanteric epiphyscodesis at 6-8 years of age to prevent progressive varus deformity has also been suggested[21,41], but if varus already exists at presentation without much shortening, then advancement of greater trochanter can restore effective abductor mechanism and can prevent trochanteric impingement during abduction[17,21,42].

Progressive coxa valga with lateral subluxation and acetabular dysplasia (Choi Type II B)

Progressive lateral subluxation can be detected by deterioration of centre –edge angle. Such cases can be managed by: Varus osteotomy of proximal femur with or without extension osteotomy, or epiphyseodesis of medial portion of capital femoral physis performed concurrently[10]. Joint stability determined intraoperatively by Barlow maneuver after the procedure is the major determining factor as to whether pelvic osteotomy should be done to increase containment. Age should not be the major factor in this respect[37]. If needed, appropriate pelvic osteotomy should be done concurrently with the above procedures to improve the chances of long term success[10].

Coxa vara / valga with severe anteversion retroversion (Choi Type III A)

These complications can be managed by appropriate corrective osteotomy and if needed, acetabular containment procedure concurrently or secondarily[10]. Epiphyseodesis of contra lateral distal femur can be done to prevent a projected limb length discrepancy[10]. Recently, a vertical 'L' type osteotomy of proximal femur has been described which can provide elongation of femoral neck, biomechanical improvement of the joint, congruity of femoral head and equalization of leg length discrepancy[43].

Pseudoarthrosis of the neck with pathological slipping of capital femoral epiphysis (Choi Type III B)

Such cases, although infrequently reported[10,35], present one of the most difficult problems to deal with and are most unpredictable regarding the outcome of treatment. The reported modalities of treatment include: bone grafting alone (reserved for cases with large and viable head only[35,44]), bone grafting with subtrochanteric valgus osteotomy[10], vascularised iliac bone graft[45] and surgical conversion[35] to Choi Type IV B, so that they may be treated by trochanteric arthroplasty and other adjuvant procedures. Besides this, other associated problems like altered acetabular development, limb length discrepancy, relative trochanteric overgrowth etc. need also be managed accordingly. Despite of all attempts at reconstruction, these cases often fare poorly.

Destruction of femoral head with small medial remnant of neck (Choi Type IV A)

These cases are best treated like Choi Type IV B (complete loss of femoral head and neck with no articulation of hip[10]). Other options include either a Chiari or valgus osteotomy or both, along with distal trochanteric advancement[35]. It has suggested that a varus osteotomy for stabilization should probably be avoided because of further varization effect of the trochanteric apophysis with absent femoral epiphysis[35].

Pathological dislocation of hip joint

If the femoral head is reasonably intact, early open reduction and stabilization is required because absence of physiologic load-bearing for bone growth and progressive soft tissue contracture will increase the deformity and make further treatment more difficult[37]. However, late attempts at open reduction usually yield poor results and are often complicated by redislocation and pain[21]. If the head and / or neck is grossly destroyed or absent, such cases should be treated like Choi Type IV B (below).

Complete loss of femoral head and neck with no articulation of hip (Choi Type IV B)

This is the most frequently encountered group amongst the sequelae of septic hip in infancy[10,35,37]. Here, goals of treatment are multifold- to provide a substitute for the femoral head and neck, to provide stability to the reconstructed hip joint and finally to correct any deformities and limb length discrepancy. Also, provision has to be made to compensate for recurrence of deformity and / or hip instability along with skeletal growth.

Of historical importance is a 'Y' type of osteotomy of proximal femur, inserting the medial limb of 'Y' into the acetabulum[46,47]. The most common reconstructive procedure reported for this subgroup is Greater Trochanteric Arthroplasty along with adjunctive and secondary procedures to provide acetabular coverage, maintain limb alignment and correct limb length discrepancy. Arthrodesis[34] is another option if the work demands are high or as an alternative to total hip arthroplasty in the

SEQUELAE AFTER SEPTIC ARTHRITIS OF HIP IN INFANTS.

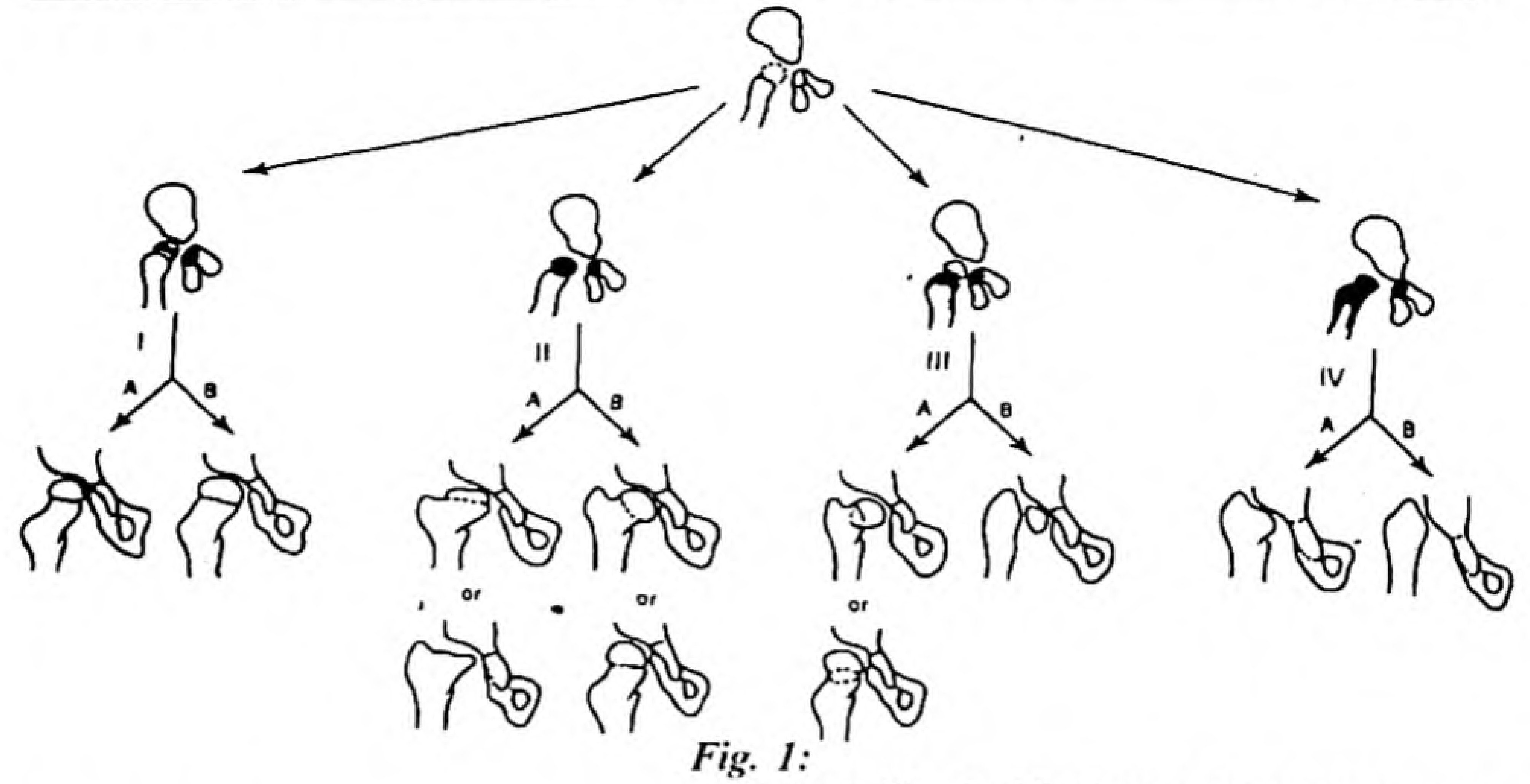

Fig. 1:

Type I A: No residual deformity;
B: Mild coxa magna.

Type II A: Coxa breva with a deformed head;
B: Progressive coxa vara or valga due to asymmetrical, premature physeal closure.

Type III A: Slipping at the femoral neck, resulting in coxa vara or valga with severe anteversion or retroversion;
B: Pseudoarthrosis of the femoral neck.

Type IV A: Destruction of the femoral head and neck, with a small medial remnant of the neck;
B: Complete loss of the femoral head and neck and no articulation of the hip. (after Choi et al).[10]

event of multiple recurrences of dislocation, painful instability and degeneration of hip joint. Recently, use of vascular-pedicled iliac crest graft to replace the destroyed femoral head and neck has been reported[48] to give good results in terms of graft incorporation, head-neck substitution with graft hypertrophy and remodeling, vertical hip stability, range of motion and pain relief. Reconstruction osteotomy of remnants of neck and / or head of femur and a callus distraction procedure in the direction of acetabulum with Kalnberz external fixation device has been reported to allow stabilization of the hip joint, new bone formation in the anatomic place of femoral head, and retention of hip joint function[19]. Further options include Excisional Arthroplasty and Total Hip Arthroplasty. However, the role of 'no treatment' still exists in selected conditions. If the lesions are bilateral, it is better to leave them untreated[32]. The long term follow-up of untreated dislocations with an absent femoral head[20,21,26,33,50] has shown that function is reasonably good with relative freedom from pain and a fair range of movement though, naturally, with a Trendelenberg gait and increased lumbar lordosis.

Limb Length Discrepancy (LLD)[21,37,40,48,51,52]

This is an inseparable part of the spectrum of sequelae of septic hip and is often the patient's major concern both cosmetically and functionally. Factors that influence LLD include: age at onset of disease, extent of destruction of hip, involvement of other joints of same / contralateral limb. One major prerequisite prior to correction of any LLD is stability of hip, otherwise lengthening process will further compromise the stability of hip due to soft tissue tension. Moreover, some of the LLD will be reduced by relocation of head itself. Various modalities of management of LLD include shoe raise (£ 2.5 cm), subtrochanteric valgus osteotomy, ipsilateral femoral / tibial lengthening by Ilizarov method, epiphyseodesis of contra lateral distal femur etc.

Greater Trochanteric Arthroplasty

This procedure involves reconstruction of hip joint by redirecting the greater trochanter into the acetabulum, thus substituting the trochanteric apophysis for the absent femoral head[10]. It was initially described for adult hips[53,54], but later became a standard procedure for paediatric hips, too[10,21,25,35,36,38,44,55-58].

Principle

It is based upon the principle that greater trochanteric apophysis undergoes remodeling into a size and shape resembling capital femoral epiphysis when it is subjected to physiological weight bearing stresses after being placed inside the acetabulum[36,38,59]. This remodeling potential may be explained by the common origin of femoral head and trochanteric apophysis from a single epiphysis[31].

Technique

Conventional technique:

Preoperative traction with or without adductor tenotomy is usually required to bring superior border of trochanter to the level of weight bearing surface of acetabulum. The surgical procedure[38] involves extensive exposure of hip joint and proximal femur by extended iliofemoral approach[35,38,55] or posterolateral approach[36], adequate soft-tissue release to allow manipulation, detachment of abductors from trochanter, acetabular clearance, and abduction of hip to place greater trochanter deeply inside the acetabulum. This is followed by distal reattachment of abductors and reattachment of rectus femoris and tensor fascia lata. Concurrent or secondary acetabular osteotomy to improve coverage of femoral head and / or proximal femoral varus osteotomy to correct limb alignment should be done[38].

Alternative technique[55]

This involves subtrochanteric osteotomy, rotation of proximal fragment through 180o around the axis of femur to place greater trochanter deeply into the acetabulum and fixation with intramedullary nail with limb in weight bearing position.

Ideal age for surgery

This is a controversial issue. Some authors[35,44,46] have recommended that it be performed when the child is as young as 2 years old to allow development of an improved pattern of gait and to permit the surgeon to monitor the limb length more reliably. On the other hand, Freeland et al[38] have reported best results in patients who were operated on at or near the time of skeleletal maturity.

Results and complications

Short term results are usually good in regards to hip articulation, remodeling, decreased limb length discrepancy, improved range of motion, pain relief and improvement in gait[10,21,35,36,38], but the long term complications include gradual subluxation of hip[38] with time ending up in frank dislocation by the age of skeletal maturity, reduction of varus necessitating a repeat varus osteotomy, degeneration, pain, shortening, limp and spontaneous ankylosis. These complications can be reduced by adjuvant and secondary procedures like proximal femoral varus osteotomy and acetabular coverage procedures[10,38].

This procedure, at best, is a salvage procedure which if done and followed meticulously, can delay the need for definitive surgery till the age of skeletal maturity.

Excisional Arthroplasty[60-64]

This involves excision of head and neck up to the intertrochanteric line. Advantages include removal of all diseased and dead tissue, relief from pain and good range of motion. Disadvantages include instability, shortening (average 3.5 cm) and limp.

Total Hip Arthroplasty[65-71]

This procedure appears promising as one of the definitive procedures for sequelae of septic hips once skeletal maturity is attained. Numerous advancements have been made in the knowledge of pathological anatomy of deformed and hypoplastic proximal femur and acetabulum along with improvement in component designs and techniques for dealing with various surgical and mechanical problems. Despite of all this, complications do occur substantially in the form of implant loosening, implant failure, redislocation, fractures, recurrence of infection etc. The major factors responsible for complications include extensive scarring due to previous infection and surgeries, wasting and inadequate healthy soft-tissue cover, malalignment and component mismatch in a deformed skeletal environment and active work demands of young adults. Still, there is a long way to go to improve the longevity of implants along with modification in available component designs so as to provide the much needed modularity in order to suit the complex anatomy of previously septic hips and to safely allow for squatting habits especially in developing countries.

References

1. Smith T. On the acute arthritis of infants. St. Bartholomew's Hospital Reports. 1874; 10: 189.
2. Phemister DB. Effect of pressure on articular surfaces in pyogenic and tuberculous arthritides and its bearing on treatment. Ann Surg. 1924; 80: 481-500.
3. Keefer CS, Holmes WF, Myers WK. The inhibition of tryptic digestion of cartilage by synovial fluid from patients with various types of arthritis. J Clin Invest.1935; 14: 131-5.
4. Curtiss PH, Jr. Cartilage damage in septic arthritis. Clin Orthop. 1969; 64: 87-90.
5. Nagel DA, Albright JA, Hollingsworth JR. Studies on the pathophysiology and some host defense factors in staphylococcal arthritis in the rabbit and on the relationship of septic inflammation to infection rate. Yale J Biol Med. 1966; 39: 119-28.
6. De Duve C. Lysosomes, a new group of cytoplasmic particles. *In* Subcellular Particles. 128-59, New York, Ronald Press, 1959.
7. Fell HB, Dingle JT. Studies on the mode of action of vitamine A: Lysosomal protease and the degradation of cartilage matrix. Biochem J. 1963; 87: 403-8.
8. Lunseth PA, Heiple KG: Prognosis in septic arthritis of the hip in children. Clin Orthop. 1979; 139: 81-5.
9. Wilesi JJ, Eraser GA. Septic arthritis in childhood. Can J Surg. 1979; 22:326.
10. Choi IH, Pizzutillo PD, Bowen JR, Dragann R, Malhis T. Sequelae and reconstruction after septic arthritis of hip in infants. J Bone Joint Surg. 1990 Sep; 72(8)-A: 1150-65.
11. Griffin PP, Green WT. Sr. Hip joint infections in infants and children. Orthop Clin North Am. 1978; 9: 123-34.
12. Bennett OM, Namnyak SS. Acute septic arthritis of the hip joint in infancy and childhood. Clin Orthop. 1992; 281: 123-32.
13. Kuo KN, Lloyd-Roberts GC, Orme IM and Sopthall JE. Immunodeficiency and infantile bone and joint infection. Arch Dis Child. 1975; 50: 51.

14. Ogden JA. Changing patterns of proximal femoral vascularity. J Bone Joint Surg. 1974 Jul; 56-A: 941-50.
15. Trueta J. The normal vascular anatomy of the human femoral head during growth. J Bone Joint Surg. 1968; 39-B (2): 358-94.
16. Chen CE, Ko JY, Li CC, Wang CJ. Acute septic arthritis of the hip in children. Arch Orthop Trauma Surg. 2001; 121: 521-6.
17. Eyre-Brook AL. Septic arthritis of the hip and osteomyelitis of the upper end of the femur in infants. J Bone Joint Surg. 1960; 42-B (1): 11-20.
18. Murray BE, Branco AJ, Rhodes KH. Septic arthritis in children. Orthop Clin North Am. 1975; 6: 923.
19. Murray BE, Branco AJ, Rhodes KH. Septic arthritis of hip in children. J Bone Joint Surg. 1976; 58-A: 388.
20. Fabry G, Meire E. Septic arthritis of the hip in children. Poor results after late and inadequate treatment. J Pediatr Orthop. 1983; 3: 461-6.
21. Hallel T, Salvati EA. Septic arthritis of the hip in infancy. End result study. Clin Orthop. 1978; 132: 115-28.
22. Little DG, Barrett IR. Septic arthritis of hip in infancy. Aust NZ J Surg. 1993; 63: 116-9.
23. Nade S. Acute septic arthritis in infancy and childhood. J Bone Joint Surg. 1983; 65-B:234-41.
24. Paterson DC. Acute suppurative arthritis in infancy and childhood. J Bone Joint Surg. 1970; 52-B: 474-82.
25. Stetson JW, Deponte RJ, Southwick WP. Acute septic arthritis of the hip in children. Clin Orthop. 1968; 56: 105-16.
26. Wopperer JM, White JJ, Gillespie R, Obletz BE. Long-term follow up of infantile hip sepsis. J Pediatr Orthop. 1988; 8(3): 322-5.
27. Lack CH. Chondrolysis in arthritis. J Bone Joint Surg. 1959; 41-B: 384.
28. Lack CH. Chondrolysis. Ann Phys Med. 1961; 6: 93-9.
29. Wientroub S, Lloyd-Roberts GC, Fraser M. The prognostic significance of the triradiate cartilage in suppurative arthritis of the hip in infancy and early childhood. J Bone Joint Surg. 1981; 63-B (2): 190-3.
30. Borella L, Goodbar JT, Summitt RL, Clark GM. Septic arthritis in childhood. J Pediatr. 1963; 62: 742.
31. Lloyd-Roberts GC. Suppurative arthritis of infancy. Some observations about prognosis and management. J Bone Joint Surg. 1960; 42-B (4): 706-20.
32. Sharrard WJW. Paediatric orthopaedics and fractures. Blackwell Scientific Publications, Oxford. 1993; 3rd Ed. Vol 2: 1241-6.
33. Betz RR, Cooperman DR, Wopperer JM, Sutherland RD, White JJ, Schaaf HW et al. Late sequelae of septic arthritis of hip in infancy and childhood. J Pediatr Orthop. 1990; 10: 365-72.
34. Bryson AF. Treatment of pathological dislocation of the hip joint after suppurative arthritis in infants. J Bone Joint Surg. 1948; 30-B(3): 449-53.
35. Hunka L, Said SE, Mackenzie DA, Roagla EJ, Cruess RL. Classification and surgical management of the severe sequelae of septic hips in children. Clin Orthop. 1982; 171: 30-6.
36. Weissman SL. Transplantation of the trochanteric epiphysis into the acetabulum after septic arthritis of the hip. Report of a case. J Bone Joint Surg. 1967 Dec; 49-A: 1647-51.
37. Chang CH, Huang SC. Reconstruction for sequelae of septic hip in children. J Formos Med Assoc. 1997; 96(5): 353-8.
38. Freeland AE, Sullivan DJ, Westin GW. Greater trochanteric hip arthroplasty in children with loss of he femoral head. J Bone Joint Surg Ser A. 1980; 62:1351-61
39. Hasler CC, Morscher EW. Femoral neck lengthening osteotomy after growth disturbance of proximal femur. J Pediatr Orthop. 1999 Oct; 8 (4): 271-5.
40. Harper MC, Canale ST, Cobb RM. Proximal femoral osteotomy: a trigonometric analysis of effect on leg length. J Pediatr Orthop. 1983; 3: 431-4.
41. Langenskiold A, Salenius P. Epiphyseodesis of the greater trochanter. Acta Orthop Scand. 1967; 38: 199-219.
42. Doudoulakis JK. Trochanteric advancement for premature arrest of the femoral - head growth plate. 6-year review of 30 hips. Acta Orthop Scand. 1991 Apr; 62 (2): 92-4.
43. Papavasiliou VA, Kirkos JM. Reconsruction of residual deformities of the hip. A new surgical option. Clin Orthop. 1997 Aug; (341): 123-7.
44. Lloyd-Roberts GC. Septic arthritis in infancy. Internat Orthop. 1978; 2: 97-100.
45. Hou SM, Hang YS, Liu TK. Ununited femoral neck fractures by open reduction and vascularised iliac bone graft. Clin Orthop. 1993 Sep; 294: 176-80.
46. Harmon PH. Surgical treatment of the residual deformity from suppurative arthritis of the hip occurring in young children. J Bone Joint Surg. 1942 Jul; 24-A: 576-85.
47. L' Episcopo JB. Stabilization of pathological dislocation of the hip in children. J Bone Joint Surg. 1936 Jul; 18-A: 737-42.
48. Cheng JC, Aguilar J, Leung PC. Hip reconstruction for femoral head loss from septic arthritis in children. A preliminary report. Clin Orthop. 1995 May; (314): 214-24.
49. Krumins M, Kalnins J, Lacis G. Reconstruction of the proximal end of the femur after hematogenous osteomyelitis. J Pediatr Orthop. 1993; 13: 63-7.
50. Obletz BE. Acute suppurative arthritis of the hip in neonatal period. J Bone Joint Surg. 1960; 42-A: 23-30.
51. Cheng JC, Lam TP. Femoral lengthening after Type IV B septic arthritis of the hip in children. J Pediatr Orthop. 1996 Jul-Aug; 16 (4): 533-9.
52. Wang CT, Huang SC. The Ilizarov technique for treatment of sequelae of childhood acquired bone and joint infection. J Formos Med Assoc. 1999 Mar; 98 (3): 175-82.
53. Colonna PC. A new type of reconstruction operation for old ununited fracture of the neck of the femur. J Bone Joint Surg. 1935 Jan; 17-A: 110-22.
54. Wilson PD. Trochanteric arthroplasty in the treatment of ununited fractures of neck of femur. J bone Joint Surg. 1947 Apr; 29: 313-58.
55. Axer A, Aner A. A new technique for greater trochanteric hip arthroplasty. J Bone Joint Surg.1984 May; 66-B: 331-3.
56. Dal Monte A, Capello A, Doncelli O. Trochanteroplasty in the treatment of infantile septic arthritis of the hip. J Orthop Traumatol. 1984; 00: 145-52.
57. Lloyd-Roberts GC. Some aspects of orthopaedic surgery in childhood. Ann Roy Coll Surg. 1975; 57: 25-32.
58. Tachdjian MO. Paediatric Orthopaedics, Philadelphia, W.B. Saunders Co. 674, 1972.
59. Morgan JD, Somerville EW. Normal and abnormal growth at the upper end of the femur. J Bone Joint Surg. 1960 May; 42-B: 264-72.
60. Collis DK, Johnston RC. Complete femoral-head and neck resection. J Bone Joint Surg. 1971; 53-A: 296-7.
61. Haw CS, Gray DH. Excision arthroplasty of the hip. J Bone Joint Surg.1976; 58-B: 44-7.
62. Milgram JW, Rana NA. Resection arthroplasty for septic arthritis of the hip in ambulatory and nonambulatory adult patients. Clin Orthop. 1991; 272; 181-91.
63. Nelson CL. Femoral head and neck excision arthroplasty. Orthop

Clin North Am. 1971; 2: 127-37.
64. Tuli SM, Mukherjee SK. Excision arthroplasty for tuberculosis and pyogenic arthritis of the hip. J Bone Joint Surg.1981; 63-B: 29-32.
65. Cherney DL, Amstutz HC. Total hip replacement in the previously septic hip. J Bone Joint Surg .1983; 65-A: 1256-65.
66. Dudkiewicz I, Salai M, Chechik A, Ganel A. Total hip arthroplasty after childhood septic hip in patients younger than 25 years of age. J Pediatr Orthop. 2000; 20(5): 585-7.
67. Dunn HK, Hess WE. Total hip reconstruction in chronically dislocated hips. J Bone Joint Surg. 1976; 58-A: 838-45.
68. Jupiter JB, Karchmer AV, Lowell JD, Harris WH. Total hip arthroplasty in the treatment of adult hips with current or quiescent sepsis. J Bone Joint Surg. 1981; 63-A: 194-200.
69. Woolson ST, Harris WH. Complex total hip replacement for dysplastic or hypoplastic hips using miniature or microminiature components. J Bone Joint Surg. 1983; 65-A: 1099-108.
70. Kim YH, Han DY, Park BM. Total hip arthroplasty for tuberculous coxarthrosis. J Bone Joint Surg. 1987; 69-A: 718-27.
71. Kim YH. Total arthroplasty of the hip after childhood sepsis. Br Ed Soc Bone Joint Surg. 1991; 73-B: 783-6.

TOXIC MEGACOLON: A REVIEW

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Introduction

Toxic megacolon is a rare but potentially fatal complication of pseudomembranous colitis. Toxic dilatation complicating pseudomembranous colitis was first described in 1968 (1). This complication is usually seen in association with idiopathic inflammatory bowel disease or infectious colitis and is characterized by segmental or diffuse dilatation of the colon of 6 cm or more (2-4). Systemic toxicity and non-obstructive colonic dilatation in the setting of acute inflammatory colitis are quintessential features of this disorder (1-4).

Incidence

The exact incidence of toxic megacolon is not known. The life time risk of developing toxic megacolon in inflammatory bowel disease was estimated to be 1-5% twenty years ago but is likely to have decreased with early recognition and better management of this complication (4, 5). Clinically significant C. difficile colitis occurs in 1% of patients admitted to the hospital and a small number of them develop toxic megacolon (6). In a recent outbreak of C. difficile colitis in Dublin 5 out of 138 patients with C. difficile colitis developed severe fulminant colitis (7).

Etiology

The etiology of toxic megacolon is diverse (Table 1). Toxic megacolon is most commonly associated with inflammatory bowel disease but may also occur as a complication of infectious colitis, ischemic colitis, and obstructive colon cancer (4). Toxic megacolon from infectious colitis is uncommon. Cytomegalovirus infection is the main cause of toxic megacolon in patients with AIDS and may also precipitate toxic megacolon in inflammatory bowel disease.

Pathogenesis

The exact mechanism of dilatation of the colon in various conditions associated with toxic megacolon is not known. Caprilli et al (8) have proposed overproduction of soluble inflammatory mediators as a pathogenic mechanism. They believe these mediators may elicit inhibitory extrinsic visceral reflexes that inhibit smooth muscle tone. There is some intriguing evidence that Nitric oxide may be involved in the pathogenesis of this disorder. Nitric oxide is a strong inhibitor of smooth muscle tone and is generated in macrophages and smooth muscle cells of an inflamed colon. Mourelle et al (9), studied inducible nitric oxide in surgical colonic specimens from patients with toxic megacolon , patients with severe non-toxic ulcerative colitis and non-obstructive colon cancer. The synthetic activity and the amount of inducible nitric oxide were increased in the muscular layer of patients with toxic megacolon as compared to patients with colitis and colon cancer. In animal studies bowel decontamination by non-absorbable oral antibiotics leads to decrease in inducible nitric oxide synthesis suggesting a role for bacterial products (10).

Amelioration of dilatation with nitric oxide synthase inhibitors in animal studies lends support to the nitric oxide hypothesis and may suggest a role of these compounds in the treatment of toxic megacolon in future (10). Although damage to the myenteric plexus has been suggested as contributing factor, it does not play a significant role in most patients (11). Hypokalemia and other electrolyte disturbances also do not contribute to dilatation in the majority of patients. Opiates and antidiarrheals may worsen colonic dilatation by inhibiting smooth muscle tone.

Pathology

Pathologically toxic megacolon is characterized by severe inflammation extending deep through all the layers of colon with infiltration by neutrophils, lynphocytes, histiocytes and plasma cells. Myocyte degeneration and necrosis is seen early in the course followed by granulation tissue formation. The submucosal and myenteric nerve plexuses are generally preserved suggesting limited , if any, role of neuropathic mechanisms in the process of toxic dilatation (11). The depth of inflammation seems to correlate with the extent of dilatation.

Clinical features

Toxic megacolon occurs early in the course of pseudomembranous colitis and may be the initial presentation in some cases (12). It can occur in males and

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females of all age groups. Patients with idiopathic inflammatory bowel disease (IBD) are at risk of this complication early in the course of their disease and sometimes toxic megacolon may be the initial presentation (3). In IBD patients it is more common in ulcerative colitis than Crohn's disease. Toxic dilatation is more likely to complicate pancolitis than segmental disease. Patients typically have severe acute colitis with bloody diarrhea, which is resistant to conventional therapy. Alleviation of diarrhea may be an early clue to the toxic dilatation. Precipitating factors like hypokalemia and use of anticholinergic medications and opiates may be identified in some patients. Inordinate distention from a barium enema or colonoscopy may further compromise blood supply and facilitate mucosal uptake of bacterial products contributing to toxic dilatation. Discontinuation or rapid tapering of corticosteroids and sulphasalazine or mesalamine may trigger the process of progressive dilatation in IBD patients.

Table 1. Etiology of toxic megacolon

A. Idiopathic inflammatory bowel disease
  Ulcerative Colitis
  Crohn's Disease

B. Infectious colitis
  a. Bacterial
    Clostridium difficile
    Shigella
    Salmonella
    Campylobacter
    Yersinia
    Aeromonas
  b. Parasitic
    Amoeba
    Cryptosporidium
  c. Viral
    Cytomegalovirus
    Culture negative colitis
  d. Drugs
    Methotrexate
    Gold
  e. Miscellaneous
    Ischemia
    Kaposi's sarcoma

On physical examination patients are generally very sick with altered consciousness, fever tachycardia and postural hypotension. The abdomen is usually markedly distended with tenderness. Increased tympany is typically noted in the middle and left upper quadrant due to accumulation of gas in the most anterior part of colon i.e. transverse colon. Loss of liver dullness on percussion of the right upper quadrant may be the first clinical clue to colonic perforation. Signs of localized or generalized peritonitis may be present. Steroids and analgeics may mask the symptoms and signs of toxic dilatation, making a high index of suspicion paramount to the recognition of this complication. The criteria proposed by Jalan et al (3) are still widely used for the clinical diagnosis of toxic megacolon and are summarized in Table 2.

Table 2. Diagnostic criteria for toxic megacolon

a. Radiological evidence of colonic dilatation
b. At least 3 of the following:
  Fever >38°C (101.5°F)
  Heart rate >120 /min
  Leukocytosis >10.5, 000/mm^3
  Anemia
c. At least one of the following (*in addition to the above*)
  Dehydration
  Altered consciousness
  Electrolyte disturbances
  Hypotension

Diagnosis

The diagnosis of toxic megacolon must be considered in all patients with acute or chronic diarrhea and abdominal distention. History of IBD, recent antibiotic use or recent travel may suggest the underlying etiology. The laboratory studies usually reveal anemia, leukocytosis and an elevated erythrocyte sedimentation rate or C-reactive protein concentration. Electrolyte disturbances are common. Metabolic alkalosis due to volume depletion and hypokalemia is associated with a poor prognosis (13). Hypoalbuminemia of < 30 g/L is present in about 70% of patients in later stages of the disease.

Plain radiography of the abdomen is essential for the diagnosis and day to day monitoring of patients with toxic megacolon. Dilatation of > 6 cm is frequently seen but overall clinical picture rather than the diameter of colon are important for the diagnosis. The maximum colonic diameter may be as high as 15 cm (2). Right colon and transverse colon are usually more dilated than descending colon and sigmoid colon and rectum are rarely dilated. The colonic haustral pattern is usually lost or impaired and multiple air-fluid levels are present. Deep mucosal ulcerations and marked mucosal thickening may be evident. Increased small bowel gas may be an early sign of toxic megacolon and is believed to be related to electrolyte and metabolic disturbances. High resolution ultrasonography may be used to assess the activity and response to therapy in severe ulcerative colitis and may be useful in early detection of toxic megacolon (14).

CT scan may be useful in identifying the etiology of toxic megacolon, especially in patients with AIDS who may have multiple intra-abdominal processes causing abdominal distention.

Endoscopy may provide valuable information, particularly in patients with toxic megacolon of infectious etiology. Traditionally a limited endoscopic examination has been used to obtain biopsies for fear of precipitating a perforation. However, carefully performed colonoscopy may be invaluable and has been used safely in diagnosis and management of toxic megacolon (15). Recto-sigmoid sparing is well known in pseudomembranous colitis and in 20-30% cases the entire left side of colon may be spared (12, 15, 16). Colonoscopy would be useful in making a diagnosis in such patients. Colonoscopy may also provides the opportunity for therapeutic decompression and obviate the need for surgery (12).

Treatment

The initial treatment of toxic megacolon is medical and is successful in 50% of patients. However an early surgical consultation is essential. The patient should be closely monitored in an intensive care unit and assessed daily by both medical and surgical teams. Anemia, dehydration and electrolyte abnormalities should be treated aggressively. Plain films of the abdomen should be reviewed every 12 hours initially, and daily as the patient improves. Complete bowel rest is essential and nasogastric suction may be required. All antimotility agents, narcotics and anticholinergics should be stopped. Prophylaxis for gastric stress ulceration and deep venous thrombosis are generally recommended. Broad spectrum antibiotics are recommended to lower the risk of septic complications.

Positional therapy by intermittent knee-elbow positioning and rolling is simple and has been reported to be useful in facilitating colonic gas expulsion (17, 18). Specific therapy of the underlying condition is essential. In patients with C. difficile colitis, this involves stopping the offending antibiotic and giving intravenous metronidazole and vancomycin by a nasogastric tube. In patients with inflammatory bowel disease intravenous corticosteroids are the mainstay of treatment. Cyclosporine may be useful in treatment of severe acute colitis refractory to steroid therapy, but experience with this agent in toxic megacolon is limited (19). Colectomy is generally reserved for patients who do not respond to an adequate trial of medical therapy for 48- 72 hours. Other indications for surgery include perforation and massive hemorrhage.

Prognosis

The prognosis in toxic megacolon is variable and depends largely on the underlying diagnosis. The overall mortality of toxic megacolon associated with pseudomembranous colitis between 1968 and 1992 was 31- 35% (6). Mortality was 42% in surgically treated patients and 18% in medically treated patients (6). In the recent literature the overall mortality has been reported to be 64-67% and mortality in surgically treated patients 71-100% (6).

The mortality rates in IBD patients have decreased from as high as 27% to 0-2% in the last two decades (4). The improved mortality is consequent to early recognition, better intensive care and early surgical treatment when indicated (4).

References

1. Brown CH, Ferrante WA, David WD. Toxic dilatation of the colon complicating pseudomembranous enterocolitis. Dig Dis Sci 1968; 13: 813-21.
2. Fazio VW. Toxic Megacolon in ulcerative colitis and Crohn's colitis. Clin Gastroenterol 1980; 9: 389-407.
3. Jalan KN, Circus W, Cord WI, et al. An experience with ulcerative colitis: toxic dilatation in 55 cases. Gastroenterology 1969; 57: 68- 82.
4. Sheth SG, LaMont JT. Toxic Megacolon. Lancet 1998; 351: 509- 13.
5. Grieco MB, Bordan DL, Geiss MD, et al. Toxic megacolon complicating Crohn's disease. Ann Surg 1980; 191: 75-80.
6. Trudel JL, Deschenes M, Maynard M et al. Toxic megacolon complicating pseudomembranous enterocolitis. Dis Colon Rectum 1995; 10: 1033-38.
7. Synnott K, Mealy K, Merry C, et al. Timing of surgery for fulminating pseudomembranous colitis. Brit J Surg 1998; 85; 229-31.
8. Caprilli R, Onori L. Pathogenesis of gastrointestinal distension in severe ulcerative colitis: a hypothesis. Gastroenterol Int 1992; 5: 268- 72.
9. Mourelle M, Casellas F, Guarner F, et al. Induction of nitric oxide synthase in colonic smooth muscle from patients with toxic megacolon. Gastroenterology 1995; 109; 1497- 1502.
10. Mourelle M, Vilaseca J, Guarner F, et al. Toxic dilatation of colon in a rat model of colitis is linked to an inducible form of nitric oxide synthase. Am J Physiol 1996; 33: G425- 30.
11. Norland CC, Kirsner JB. Toxic dilatation of colon: Etiology, treatment and prognosis in 42 patients. Medicine 1969; 48: 229-50.
12. Triadafilopoulos G,Hallstone AE. Acute abdomen as the first presentation of pseudomembranous colitis. Gastroenterology 1991; 101: 685-91.
13. Caprilli R, Vernia P, Colaneri O, et al. Blood pH: a test for assessment of severity in proctocolitis. Gut 1976; 17: 763- 9.
14. Arienti V, Campieri M, Boriani L, et al. Management of severe ulcerative colitis with the help of high resolution ultrasonography. Am J Gastroenterol 1996; 91: 2163- 9.
15. Alemayehu g, Jarnerot G. Colonoscopy during an attack of severe ulcerative colitis is a safe procedure and of great value in clinical decision making. Am J Gastroenterol 1991; 86: 187- 90.
16. Rubesin SE, Levine MS, Glick SN, et al. Pseudomembranous colitis with rectal sparing on barium studies. Radiology 1989; 170: 811.
17. Present DH, Wolfson D, Gelernt IM, et al. Medical decompression of toxic megacolon by "rolling": a new technique of decompression with favorable long term follow up. J Clin Gastroenterol 1988; 10: 485- 90.
18. Panos MZ, Wood MJ, Asquith P. Toxic megacolon: the knee-elbow position relieves bowel distension. Gut 1993; 34; 1726- 7.
19. Hanauer S. Inflammatory Bowel Disease. N Eng J Med 1996; 334: 841- 8.

NEPHRONOPHTHISIS

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Nephronophthisis (NPH) and medullary cystic disease (MCD) represent a disease complex heterogeneity, but have also been discussed as separate entities. This complex was Ist described by Fanconi in 1951 and fewer than 300 cases have been reported by 1980. NPH is responsible for at least 10% of renal failure in children and NPH and MCD combined together account for 4% of all hereditary renal disoorders.[1]

Nephronophthisis is a disease characterized by a chronic diffuse interstitial nephritis with few or no inflammatory cells and the presence of cysts in the medulla. The presence of these cysts has given rise to the term 'Medullary cystic disease in American Literature[2] whereas Fanconi et al emphasised the atrophy of renal tubules and parenchyma and proposed for the term 'Nephronophthisis[3]. However, both these diseases are altogether called as 'Familial Interstitial nephritis. NPH appears to be an autosomal recessive condition (Sporadic in 20%) and can be Juvenile (FJN); Infantile (FIN); or Adolescent (FAN); with an aveage age of onset of ESRD of 13(7-25) yr; 1-3yr and 19(11-28)yr, respectively[4]. Males & females equally affected. MCD appears to be an autosomal dominant and / or sporadic with an average age of onset of terminal renal failure in adulthood. Two variants with median onset of ESRD at 62yr. and 32yrs have been documented in this variety. Clinical and histopathological characteristics of both the FJN and MCD are similar with an exception of infantile form[5]. FJN is more commonly associated with extra renal involvement including retinal defects, skeletal abnormalities, aminoaciduria, hepatic fibrosis and lesions of central nervous system.

Clinical Features

These include Polyuria and Polydipsia (80%); anemia and weaknes(60%); growth retardation (40%); Azotemia (75%) and /or hypertension (20%). Urinary findings are scant with mild Proteinuria (less than 1gm/24 hrs.) and occasional red blood cells or white blood cells. Urinary concentrating defect is present early and is out of proportion to the level of renal insufficiency. Many of the patients demonstrate salt wasting which may account for the lw incidence of hypertension. Hyperkalemia, Hypokalemia, Proximal tubular acidosis, distal tubular acidosis and aldosterone resistance have been reported. Flank pain, gross haematuria or renal calculi are uncommonn in this disorder.

Pathology[6]

Kidneys appear pale red and contracted with smooth finely granular surface, thin cortex without papillary necrosis or changes in renal pelvis. Renal weight is reduced to less than 50%. Medullary cysts vary from 1mm - 1cm however papillary and cortical cysts are less common. 25% are acystic form of the disease. Cysts are lined by flattened, cuboidal epithelial cells suggestive of collectinng ducts, which contain granular eosinophilic proteinaceous material. There are decreased number of glomeruli with periglomerular fibrosis, interstitial fibrosis and inflammation. Tubules demonstrate atrophy with areas of dilatation and thickening of basement membrane and changes are out of proportion to glomerular dystrophy. Electron microscopy shows alterations in glomerular basement membrane and type II collagen as found in Alport syndrome suggesting possible faulty develpment of basement membrane in this disorder as well.

Diagnosis:

Genetics:

NPH is associated with chromosome 2[7] while MCD is associated with chromosome 1 and 6[8]. These is extensive genetic heterogeneity between the two groups.

Three are 3 different loci for NPH

- NPHP 1 (Juvenile Nephronophthisis)
- NPHP 2 (Juvenile Nephronophthisis)
- NPHP 3 (Juvenile Nephronophthisis)

Juvenile nephrnophthisis (NPHP 1), in addition can be associated with extra renal organ involvement. Gene (NPH 1) for Juvenile Nephronophthisis has been identified by positional cloning and its gene product, Nephrocystin is a novel protein of unknown function a Src-homology 3 domain. It is hypothesized that pathogenesis of NPH might be related to signaling process at focal adhesion and/or adherent junctions because most src-homology 3-containing proteins are part of focal adhesion signaling complexes.

Similarly there are two different loci for MCKD

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- MCKD 1
- MCKD 2

MCKD 1 & - MCKD 2 are associated with hyperuricemia & gout.

Ultrasound and /or CT -Scan

UsG and/or CT-Scan of Abdomen reveals small medullary Cysts (2-10mm) with a thin cortex and poor definition of corticomedullary junction. In acystic form of the 'disease, small size of the kidneys with reduced corticomedullary junction is found only[9].

Intravenous Pyelography

IVP demonstrates the thinned cortex with inhomogenous streaking restricted to the medulla and lucent enlarged medullary pyramids Accumulation of contrast medium form rings in pyramid and is pathognomonic of the disease.

Renal Biopsy

Renal Biopsy with medullary tissue is necessary to confirm the diagnosis. However, because of absence of cysts in 20% of patients, one must rely on other findings characteristic of the disorder such as family history and clinical features of the disease to make a diagnosis.

Thus for all practical purposes, the presence of renal insufficiency with marked Polyuria, in absence of haematuria or proteinuria, with a normal urinary tract and kidneys of normal or slightly reduced size occurring in patients aged from 3 to 15 or 18 years is highly suggestive of 'Nephronophthisis'.

Treatment

There is no specific treatment of the disease. However, prior counseling regarding the consanguinity of the marriage prevents the occurrence of disease. Prevention of infection, Adequate hydration, Supplementatioon with salt and bicarbonates, correction of anemia with erythropoietin, and intake of essential amino acids delays the worsening of the disease. Renal transplantation has been tried in soome cases without any recurrence of the disease in transplanted kidney. Gene therapy targeting at the various loci has been tried in some experimental animals. The process if successful, can bring a revolution in management of the disease. Overall, once the clinical manifestation develop the death occur within 5 years irrespective of time of onset of disease.

Extra Renal Manifestations

These are more commonly seen with FJN rather than other variants. Various organ involvements have been given different names as:-

Seniors syndrome (or) Senior taken synd ome[10]

This is an association of familial juvenile Nephronophthisis with abnormalities in vision. Eye involvement can be in form of tapetoretinal degeneration, retinitis pigmentosa of various origins or even simple anomalies without extinction of electroretino graphic tracings. This association is found in 40% of cases.

Mainzer - Saldino Syndroome[11]

This is an associaton of FJN with retinal degeneration resembling labers amaurosis, cone shaped epiphysis and cerebellar ataxia

Boichis Sydrome[12]

This is an association of FJN with liver fibrosis and may occur in combination with deafness, cerebellar ataxia and tapetoretinal degeneration.

Joubert Syndrome[13]

This is an association of FJN with coloboma of the optic nerve and cerebellar vermis aplasia

Jeunes Syndrome[14]

This is associatioon of FJN and retinal degeneration with asphyxiating thoracic dystrophy.

Differential Diagnosis

- Nephrogenic diabetes inspidius: Delayed onset of Polyuria after birth and abnormal sensitivity to vasopressin (ADH) differentiates NDI from FJN.
- Oligomeganephronia: Rare familial associaiton and less rapid renal failure differentiates oligomega nephronia from FJN.
- Infantile Polycystic kidney disease (IPCKD): Large size of kidney, cortical location of cyst and excretory urography showing a pattern of medullary ductal ectasia differentiates IPCKD from FJN
- Medullary sponge kidney (MSK): Different genetics and no impairment of renal function differentiates MSK from FJN.
- Glomerulocystic kidney disease (GCKD): Characteristic location of cyst on glomeruli on ultrasound examination of abdomen differentiates GCKD from FJN.

References:

1. Klernknecht C: Inheritance of Nephronophthisis In topics in Renal medicine: Inheritannce of kidney and urinnnary tract diseases, edited by Spitzer A, Avner ED, Boston, Kluwer Academic Pulbishers PP, 1989; 277-294.
2. Smith C.H and Graham, J.B. Congenital Medullary cysts of the kidneys with severe refractory anemia. Am J of disease oof children, 1945;69;370-8.
3. Fanconi, G., et al. Die familiare juvenile Nephronophthisis. Helvetica Paediatrica Acta, 1951;6:1-49.

4. Hildebrandt F, et al. Molecular genetic indetification of families with juvenile nephronoophthisis type 1: Rate of progression of renal failure APN study group. Arbeitsgemeinschaft furer Paediatrische Nephroologie. Kidney Int. 1997;51:261-9.
5. Gagnadoux MF, et al. Infantile chronic tubulo-interstitial nephritis with cortical microcysts: Variant of nephronophthisis or new disease entity? Pediatr Nephol 1989;3:50-55
6. Straun, M.B. Clinical and Pathological aspects of cystic disease of renal medulla. Annals of Internal medicine 1962;57:373-81.
7. Antignac C, et al. A gene for familial juvenile nephronophthisis (recessive medullary cytic disease) maps to chr 2p. Nat genet 1993;3:342-5.
8. Scolari F, et al. Genetic heteroogeneity for autosomal dominant medullary cystic disease (ADMCKD). J Am SOC Nephrol 1998;9:393A.
9. Blowey D, et al. Ultrasonic findings in juvenile nephronophthisis. Pediatr Nephrol 1996;10:22-24.
10. Fillartre, J.P. et al. Senior Loken Syndrome - a study of 8 cases from 5 families.. Clinical nephroology, 1976;5:14-19.
11. Manizer F, et al. Famillial nephropathy associated with retinitis pigmentosa, cerebellar ataxia and sketetal abnormality. American journal of medicine, 1970;49:556-62.
12. Boichis, H., et al. congenital hepatic fibrosis and Nephronophthisis. Quarterly journal of medicine, 1973;42:221-33.
13. Saraiva JM, Baraister M: Joubert syndrome. A review. Am J med Genet. 1992;43:726—31.
14. Popovic-Rolovic, M., et al. Juvenile nephronophthisis associated with retinal pigmentary dystrophy, cerebellar ataxia and skeletal abnormalities Archives of disease in childhood 1976;51:801-3.

SARS VIRUS : A NOVEL CORONAVIRUS

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INTRODUCTION

SARS or Severe acute respiratory syndrome is a new clinical entity caused by an infectious agent unknown to humans till now.

Previously thought to be caused by Metapneumovirus belonging to family Paramyxoviridae, it is now known, by genomic sequencing, to be a coronavirus of family Coronaviridae.

Coronaviruses are RNA viruses, which were first isolated from chickens in 1937. In the 1950's when the first Rhinoviruses were cultured, 30% of infectious agents of common cold could still not be accounted for till in 1965 Tyrell and Bynoe cultivated the first human coronavirus on human ciliated embryonal tracheal cells [1,2,3]. Hamre and Procknow isolated HCOV- 229E from a patient with an upper respiratory tract infection by serial blind passage in secondary cultures of human embryonic Kidney cells [4,5]. Most of the other human coronaviruses that were grown in this way were never successfully adapted to serial passage in continuous cell lines and are no longer available for study. HCOV- OC43 and the antigenically related OC38 strains were isolated in human fetal tracheal organ culture, and then serially passaged by intracerebral inoculation into suckling mouse brain[6]

MORPHOLOGY

Coronaviruses are large, enveloped viruses that cause highly prevalent disease in humans and domestic animals[7,8]. They have the largest genome of all RNA viruses and replicate by a unique mechanism, which results in high frequency of recombinations. Virion particles are 100-120nm in diameter and viral envelopes are studded with long petal shaped spikes, giving them the appearance of a crown (latin corona). Inside the virion is a single stranded, positive sense genomic RNA 27-32 kb in size, the largest of all RNA virus genomes. The genome associates with the N (nucleocapsid) phosphoprotein (50-60 kd) to from a long, flexible, helical nucleocapsid [9,10]. The virus core is enclosed by a lipoprotein envelope, which is formed during budding from intracellular membranes [11,12,13]. Two types of prominent spikes line the outside of the virion. The long spikes (20nm), consisting of S glycoprotein are present in all coronaviruses, the short spikes, consisting of haemagglutinin esterase glycoprotein are present in only some coronaviruses. The envelope also contains the M glycoprotein which spans the lipid bilayer three times [14,15,16]. Also present is an e - protein which is present in smaller amounts than the other proteins [17,18,19].

Coronaviruses genomic RNA is capped, polyadenylated, positive stranded RNA which function directly as mRNA [20,21]. At the 3'end and 5'end of RNA are terminal repeats for replication and transcription.

Coronaviruses mutate at a high frequency because of the error prone nature of RNA polymerase [22]. Temperature sensitive(ts) mutants have been classified into seven complementation groups [23]. Deletion type mutations also occur. A unique feature of coronavirus genetics is a high frequency of RNA recombinations which is as high as 25%, a feature rarely seen in unsegemented viruses [24]. The high frequency of RNA recombination in coronaviruses is due to a unique feature of coronaviruses RNA synthesis, which involves discontinuous transcription and polymerase jumping.It is possible that the viral polymerase associated with the incomplete nascent RNA dissociate from its template at a random point and switches to a homologous site on a different RNA template to complete RNA synthesis by a copy choice mechanism [25,26].

SARS CORONAVIRUS

The SARS coronavirus, which is presently the cause of global concern, is different from other human coronaviruses in many respects. Its genome, sequenced in a matter of three weeks, has shown only 50-60% sequence homology with other coronaviruses [27]. SARS-associated coronavirus has been seen to easily grow on many continuous cell lines like Vero E6, MDCK etc. whereas other coronaviruses are very fastidious in their growth. No other human coronavirus has been grown on Vero E6 cell lines previously and the only other coronavirus to be grown on Vero E6 cells is porcine epidemic diarrhea virus, and it also requires the addition of trypsin to culture medium for growth in Vero E6 cells. Moreover, porcine epidemic diarrhea virus adapted to vero cells results in a striking different cytopathic effect (i.e cytopalasmic vacuoles and

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days after onset of signs and symptoms whereas ELISA detects antibody after 21 days of illness.

BIBLIOGRAPHY

1. Almeida JD, Tyrell DJ. The morphology of three previously uncharacterized human respiratory viruses that grow in organ culture J Gen virol 1967; 1:175-178.
2. Bradbarne AF,Tyrell DJ.Coronaviruses of man. Prog. Med Viroll1971;13:373-403.
3. Tyrell DA, Bynoe ML. Cultivation of a novel type of common cold virus in organ culture a Prog Med J 1965;1:1467-1470.
4. Hamre D. Kindig DA, Mann J. Growth and intracellular development of a new respiratory virus J Virol 1967; 1: 810-816.
5. Hamre D, Procknow JJ. A new virus isolated from the respiratory tract. Proc Soc Exp Biol Med 1966; 121: 191-193.
6. McIntosh K, Becker WB, Channock RM. Growth in suckling mouse brain of "IBV- like" viruses from patients with upper respiratory tract diseases. Proc Natl Acad Sci 1967; 58: 2268-2273.
7. Siddell SG, Snyder EJ. Coronaviruses, toraviruses and arteroviruses in collier Leds. Topley and Wilson Microbiology and microbial infections London Edward Acnold 1998; 1; 463-484.
8. Wege H, Sidelell S, ter menlers V. The biology and pathogenesis of coronaviruses Curr top Microbial immunol 1982;99:165-200.
9. MacNaughton MR, Davies HA, Nermut MV. Ribonucleo protein like structures from coronaviruses particles. J Gen Virol 1978; 39: 545-549.
10. Sturman LS, Holmes KV, Behnke J. Isolation of coronavirus envelope glycoprotein and interaction with the viral nucleocapsid. A F. J virol 1980; 30: 449-462.
11. Griffiths G, Rottier PJM cell biology of viruses that assemble along the biosynthetic pathway Semen cell biol 1992; 3: 367-381.
12. Oslero LS, coronaviruses in Dalton AJ, Hagnenac F eds. Ultrastructure of animal viruses and Bacteriophages. An atlas New York, Academic Press, 1973; 331-343.
13. Tooze J, Tooze SA, infection of AtT20 musine pituitary tumor cells by mouse hepatitis virus strain A59; virus budding is restricted to the golgi region. Eur J cell biol 1985;37: 203-212.
14. Machamer CE, Grim MG, Esquela A et al. Retention of a Cis Golgi protein requires polar residue on face of a predicted alpha-helix in the transmembrane domain. Mol biol cell 1993; 4:695-704.
15. Machemer CE, Mantone SA, Rose JK. The E1 glycoprotein of an avain corona virus is targeted to cis Golgi complex. Proc Natl Acad Sci USA 1990; 87: 6944-6948.
16. Machamer CE, Rose JK. A Specific transmembrane domain of a coronavirus E1 glycoprotein.J Cell Biol 1987;105:1205-1214
17. Godet M. E Harridon R. Vautherot JF.TGEV coronavirusORF4 encodes a membrane protein that is incorporated into virions.Virology1992;188:666-675
18. Vennema H, Godeke GJ, Rossen JWA. Nucleocapsid independent assembly of coronavirus like particles by coexpression of viral envelop protein genes. EMBO J 1996;15:2020-2028
19. Yu X, Bi W, Weiss SR. Mouse hepatitis virus gene 5b protein is a new virion envelope protein.Virology.1994;202:1018-1023.
20. Lomniczi B.Biological properties of avian coronavirus RNA.J Gen Virol. 1977;36:531-533.
21. Schochetman B, Stevens RH, Simpson RW. Presence of infectious polyadenylated RNA in the coronavirus avian bronchitis.Virology 1977;77:772-782
22. Steinhauher DA, Holland JJ. Direct method for quantitation of extreme polymerase error frequencies at selected single base sites in viral RNA. J Virol 1986;57:219-228.
23. Lepare-Geofart I, Hingley ST, Chua MM. Altered pathogenesis of murine coronavirus MHV-A59 is associated with a Q159L aminoacid substitution in the spike protein. Virology1997;239:1-10
24. Baric RS, Schaad MC. Establishing a genetic recombination map for murine coronavirus strain A59 complimentation groups. Virology1990;177:646-654.
25. Lai MMC. RNA recombination in animal and plant viruses. Mobiol Rev 1992;56:61-69.
26. Banner LR, Keck JG, Lai MM. A clustering of RNA recombination sites adjacent to a hypervariable region of the peplomer gene of murine coronavirus. Virology 1990;175:548-555
27. World Health Organization. Acute respiratory syndrome.Wkly epidemiol Rec 2003;78:73-74.
28. Hoffman M, Wyler R. Propagation of the virus of porcine epidemic diarrhea in cell cultures. J clin microbial 1988;26:2235-2239.
29. Drosten C, Gunther S, Preiser W. Identification of a novel coronavirus in patients with severe acute respiratory syndrome,N Engl J Med 2003 Published in april 10,2003 at http://www.nejm.org/cgi/content
30. Kathryn V. Holmes.coronaviruses in David MK, Peter MH, Drane EG. Fields virology 4th ed. Lippincott Williams and Wilkins
31. Centre for Disease Control and prevention. Severe acute respiratory syndrome. Updated interim case fatality associated with SARS. Accessed 8th may 2003 at http://www.cdc.gov/ sars/ case definition.htm
32. World Health Organization. Cumulative number of reported cases (SARS) from 1st November to 14th May 2003. Accessed 14th may 2003 at http://www.who.int/cst/sars country/ 2003
33. Preliminary clinical description of SARS. MMWR morb wkly Rep 2003;52:255-256.
34. Guidelines for collection and handling of specimen for investigation of potential cases of SARS. Accessed 8th may 2003. at http://www.nicd.org/guidelines
35. Centre for Disease Control and prevention. Severe acute respiratory syndrome. Updated interim case definition. Accessed 8th may 2003 at http://www.cdc.gov/ sars/case definition.htm
36. Drosten C, Gunther S, Preiser W. Identification of a novel coronavirus in patients with severe acute respiratory syndrome,N Engl J Med 2003 Published in april 10,2003 at http://www.nejm.org/cgi/content
37. Leo LM On KW, Winsie L. Rapid diagnosis of a coronavirus associated with severe acute respiratory syndrome. Accessed 8thMay2003. http://www.clinchem.org.
38. Ksaizi TG, West CP, Pollin PE. ELISA for detection of antibodies to Ebola viruses. J Infect Dis 1999;179:192-198.

BACTERIOLOGICAL PROFILE IN ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

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Abstract: A prospective study on 120 patients of chronic obstructive pulmonary disease (COPD) was carried out to obtain the accurate information on distal microflora during their acute exacerbations.

Early morning sputum samples were collected within 24 hours of admission in sterile bottles after an antiseptic oral rinse. The samples which qualified from lower respiratory tract were subjected to culture and sensitivity.

Sixteen patients (13.33 %) grew bacteria in the order of pseudomonas in 10, staph aureus in 4 and strep. pyogens in 2 cases. 75% of culture positive patients had clinical and radiological evidence of consolidation. Organism grown were universally sensitive to levofloxcin and amikacin. 8 patients who were culture negative died. The low bacterial yield by direct sputum sampling may be increased by a bronchoscopic collection of sputum.

Key Words: COPD, Acute exacerbation, Bacteriological profile.

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Introduction

Chronic obstructive lung disease has recently been defined by American Thoracic Society as a "disease state characterized by the presence of air flow obstruction secondary to chronic bronchitis (airway disease) or emphysema (parenchymal disease). The air flow obstruction is generally progressive and may be accompanied by airway hyperreactivity and may be partially reversible. Chronic bronchitis is defined in clinical terms as a syndrome with presence of chronic cough with abnormal production of sputum for at least 3 months in each of the two consecutive years without other specific cause of cough. Emphysema on the other hand is defined pathologically as abnormal air space enlargement distal to terminal bronchioles(2,3).

Bacterial infections are the most common observable cause of acute exacerbation and death in COPD. Acute exacerbation is characterized by increase in cough, dysnoea and alteration in type and amount of sputum production. In this setting findings frequently associated with infection such as fever, leukocytosis or parenchymal infiltrates are absent. As the disease progresses the intervals between exacerbations shorten(1).

Pathogenic bacterial organisms are found in 50 to 80% of patients with COPD during exacernbations. Previously the predominant organisms believed were streptococcus pneumoniae, H. influenza, mycoplasma, morexella and pseudomonas (2-5). As per the recent studies the bacterial flora during acute exacerbation is changed from usual organisms to enterobacterial and pseudomonas aerogenosa with the increasing severity in COPD(6). Antibiotic therapy almost always remains empiric,(3) which is frequently an avoidable. With the background of little information available from Indian literature and large number of patients of COPD with or without acute exacerbation are attended at Sher-i-Kashmir Institute of Medical Sciences, Srinagar, Kashmir. On this basis this study was conducted to know the bacteriological profile of infections in COPD patients with exacerbation and design a proper antibiotic regimen, which will have a beneficial effect on the morbidity and mortality of the disease.

Material and Methods

120 diagnosed patients of COPD with acute exacerbation evidenced by increased dysponea, increased sputum volume and sputum purulence attending the department of Respiratory Medicine, SKIMS, Srinagar, Kashmir were studied over a period of two years. Each patient was included only once even if hospitalized more than one time. All the patients were subjected to routine investigations of haemogram, serum chemistry, electrocardiogram, x-ray chest PA view, arterial blood gas analysis, lung function tests and a high resolution computed tomography (HRCT) to rule out bronchiectasis.

Specific investigations included a sputum culture and sensitivity. Early morning sample of sputum from lower respiratory tract was collected within 24 hours of admission

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in sterile bottles after washing the oral cavity with antiseptic oral rinse (Benzydamine HCI) (10) to decrease the oral contramination and sample transported to laboratory within 15 minutes of collection. Gram's staining of the sputum in the area of maximal purulence was examined for polymorpholeukocytes (PMN) and epithelial cells. Sputum samples showing < 10 squamous epithelial cells and > 25 leucocytes/ low power field were subjected to culture examination (7). Sputum samples were stained with both Gram's and Zeil Neelson stain. For isolation and identification of gram +ve organism, blood agar was used and for gram negative bacteria, MacConkey agar was used. Blood agar was incubated at 37°C for 24 hours for aerobic bacteria and 37°C for 48 hours of anaerobic bacteria. Chocolate agar was incubated in candle jar with 5-10% CO_2. In case with multiple bacteria isolated in one sputum sample, the organism with the most colonies to be the predominant bacteria was considered.

Table -I: Clinical Characteristics of the Patients

Age (Mean+SD)	59.42+10.59
(Range)	(35-85)
Sex (M:F)	84:36
Smokers	74(61.7%)
Fever	32(26.7%)
Nacrosis	42(35.0%)

Data Analysis

Statistical methods used were '*t*' test and Fisher's exact test. All *p* values were calculated as two tailed. A '*p*' value of <0.05 was taken as significant.

Table -II: Laboratory Characteristics (All Patients)

TLC (Mean±SD (X10⁹/L)	9.60+3.92	Range	4.0-30.0
Leukocytosis (%)	36.7%		
PCV	54.0.:!:7.85	Range	38-78
pH	7.30.:!:0.07	Range	7.10-4.43
PCO^2	49.48±8.12	Range	33-80mm
**Acidosis	48.3%		
FVI>50<80	63.6%		
<50	36.4%		
***Consolidation	36(30%)		

**Leukocytosis = TLC (10x10⁹/L)*
*** Acidosis = pH<7.30*
****Radiological evidence of pneumonitis*

Results

The mean age of patient was 59.42:to 10.59 years with M:F ratio 2.1:1 with the maximum number of patients 46(38.3%) in the age group of 60-69 years, with no statistical difference in the number of patients in each group. Seventy four (61.7%) patients were smokers. Fever was present in 32(27.7%) and CO_2 narcosis in 42(35%) patients (Table-I)

Discussion

COPD remains among one of the commonest disease of elderly population. With the increase in life expectancy, the prevalence of COPD is increasing. Though a progressive disease, acute exacerbation can account for significant increase in the morbidity and mortality of these patients.

The role of respiratory tract infection in the onset of exacerbation in patients of COPD is nuclear. In most previous studies airway infection seems to be the first cause of decompensation and accounts for 80-90% of cases in whom triggering factor can be demonstrated[2]. The definition of an acute infective exacerbation in COPD is a major point of criticism in many of the studies dealing with that issue. Therefore, we recorded our data according to the Winnipeg criteria for exacernation[9].

Table -III: Laboratory Characteristics
Culture +ve Culture -ve Patients

	Culture-ve (n=104)	Culture +ve (n=16)	p value
TLC	9.27±2.98	11.78±7.66	0.092
PCV	54.53±7.8	50.75±7.78	0.209
pH	7.30±0.07	7.31±7.78	0.234
PCO_2	50.01±18.60	48.75±3.99	0.687
Leukocytosis	No 66(63.46%)	10(62.5%)	
	Yes38(36.53%)	6(37.5%)	.997
*Consolidation	No 80(76.92%)	4(25%)	
	Yes 24(23.07%)	12(75%)	.007
Acidosis	No 50(48.07%)	12(75%)	
	Yes 54(51.92%)	4(25%)	.149

** Statistically significant number of patients with consolidation had sputum culture +ve (P = 0.007)*

Study population consisted 120 patients of COPD as per the definition of American Thoracic Society[2,3] with acute exacerbation[24] with age of 59.42±10.5 years which is consistent with literature. We were careful to exclude from our study group all patients with other cough phelgm syndromes, such as bronchiectasis and asthma. Most of the patients 84(70%) had exacerbation during winter months.

Positive cultures of the sputa was seen in 16(13.33%) out of 120 patients. Ten (8.35%) of our patients were positive for pseudomonas aerogonosa that is consistent with the study by Griffth DE, Muzerek GH et al, have also found that traditional pathogens (pneumococcus) are declining in importance, while newer pathogens, such as pseudomonas, moraxella and legionella are becoming important[10]. Four patients (3.33%) and 2 patients (1.66%) were positive for staphycoccus aureus and streptococcus pyogens respectively, which is consistent with study by Herberty Reynolds who have found that these organisms are responsible for acute exacernation of COPD in 1-10% of patients.

Table -IV: Culture Sensitivity Table

	No. of patients	Sensitive	Resistant
Staph. aureus	4	1,2,3,4,14	5,7,10,13
Streptococus pyogenous	2	1,5,7,2,3,8	9,10
Pseudomonas auregenosa	10	2,3,4,12	4,6,11

1. Amoxicillin
2. Levofloxcin
3. Amikacin
4. Cefataxine
5. Genatamicin
6. Cefazolin
7. Erythromycin
8. Roxithromycin
9. Contrimoxazole
10. Cephalexin
11. Carbencillin
12. Tobramicin
13. Penicillin
14. Methicillin

Organisms were universally sensitive to Levofloxin and amikacin.

The need for antibiotics in the therapy of acute exacerbation of COPD remains controversial. This is primarily due to our inexact knowledge of the cause of these exacerbations and also to the differences in design and outcome in previously reported studies[7]. Twelve out of 16 patients (75%) with culture positive sputum had clinical and radiological evidence of consolidation which was statistically significant (P-0.007) and the organisms grown were universally sensitive to Levofloxcin and amikacin, with variable sensitivity to other drugs like (amoxycillin, cefazolin, erythromycin, gentamycin, cephalexin). Interestingly 4(3.33%) of our patients who were admitted with acute exacernation of COPD were positive for tubercular bacillus on acid fast staining (Zeel Neelson). This is not usually reported in other studies, and a possible explanation tothis could be the higher incidence of tuberculosis in our part of the world.

Antibiotic treatment was started after sputum collection and changed according to the sensitivity pattern. Among culture negative 8 patients died. None of patients among culture positive or AFB positive died during admission. There was no statistical significance of culture positive patients with age, sex, smoking, fever, leukocytosis, narcosis, acidosis and FEV_1.

Conclusion

Thus, it was observed that diagnosis of specific agent in causation of acute exacernation of COPD is difficult. The yield could be increased upto 50% by taking sample broncoscopically and antibiotic therapy remains empiric with first choice antibiotics among quinolones (Levofloxcin) and Amikacin.

References

1. Bartolome R Celli Clinical aspects of chronic obstructive pulmonary disease. Text book of pulmonary disease. :ippincott Raven Publishers Phildelphia 6th ed., Vol-II, 843: 1998.
2. Jean-Yuens Fagon, Jean Chastre, Jean-Louis et al. Characterization of distal bronchial microflora during acute exacerbation of chronic brollchitis. Am Rev Respir Dis 1990;12:1004-1006.
3. Jean-Yues Fagon, Jean Chastre, Jean-Louis et al. Characterization of distal bronchial microflora during acute exacerbation of chronic brollchitis. Am Rev Respir Dis 1990; 142:1004-1006.
3. Griffith De, Muzerek GH. Pneumonia in chronic obstructive lung disease. Infect Dis Clin North Am 1991;5(3): 467-484.
4. Dorea J. Acute bronchial infection in chronic obstructive pulmonary disease. Monaldi Arch Chest Dis 1995;50(5):366-371.
5. Allegra L, Konietzko N, Leophomate et al. Comparative safety and efficacy of sparfloxacin in the treatment of auete exacernation of COPD. J. Antimicrob Chemother 1997; 37(Suppl 4): 93-104.
6. Eller J, Ede S, Schaberg T, Neiderman MS, Mauch H, Lode HO. Infective exacerbations of chronic bronchitis-relation between bacteriologic etiology and lung function Chest 1998; 113(6):1542-1548.
7. American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1995; 152:78-121.
8. Medical Research Council. Definition and Classification of Chronic bronchitis for clinical and epidemiological purposes. Lancet 1965; 1:775.
9. Anthoniseq NR, Manfreda J, Warren CP, Harshfield ES, Harding GKM, Nelson A. Antibiotc therapy in exacerbations of chronic obstructive pulmonary disease. Annals of Internal Medicine 1987; 106:196-204.
10. Herbert Y Reynolds. Chronic bronchititis and acute infectious exacerbations: Principles and practice of infectious diseases. Fourth edition. Churchill Living Stone. Vol I, 608-12: 1995.
11. Brooke Nicotra, Rivera Mt Lumen JI, Wallac RJ Jr, Branchemella catarrhalis as a lower respiratory tract pathogen in patients with chronic lung disease. Arch Intern Med 1986; 194.

DUODENOGASTRIC REFLUX, AN IMPORTANT CAUSE OF POST CHOLECYSTECTOMY SYMPTOMS

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ABSTRACT: *The study was conducted in the department of General Surgery and Nuclear Medicine, Sher-i-Kashmir Institute of Medical Sciences from April 1999 to March 2000. Thirty patients with symptoms attributable to ultrasound documented gall stones were included in the study.*

Tc-99m Mebrofenin hepatobiliary scanning was used to measure duodenogastric reflux prior to cholecystectomy and also post cholecystectomy.

A significant increase in duodenogastric reflux was demonstrated in seven patients post cholecystectomy. Five from this group of 7 patients presented postoperatively with clinical picture of bile gastritis with chief complaints of burning epigastric pain and bilious vomiting.

Key words: *Cholecystectomy, reflux, duodenogastric, gastritis, dyspepsia, Tc- 99m Mebrofenin*

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INTRODUCTION

Cholecystectomy remains the commonest major abdominal operation being performed worldwide. The procedure usually results in excellent therapeutic outcome but 20-40% of patients continue to have a variety of gastrointestinal symptoms[1]. It encompasses a large number of biliary and non-biliary entities and is rarely due to the operation itself. For the successful treatment of such patients, the diagnosis of the cause of symptoms is of utmost importance. Causes such as retained CBD stones, CBD strictures, papillary tumours, cystic duct remnant, sphincter of oddi dysfunction and peptic ulcer disease can be diagnosed and treated. However, in a subset of patients further investigations will identify no obvious cause for post cholecystectomy complaints, and such patients are believed to have symptoms related to cholecystectomy[1]. The cholecystectomy is accompanied by an alkaline shift in gastric pH profiles that is more marked in symptomatic patients. There is much experimental and clinical evidence that gall bladder disease and cholecystectomy can increase duodenogastric reflux[2-6]. It is widely recognized that duodenogastric reflux is an important factor in the pathogenesis or symptomatology of several upper gastrointestinal tract disorders including gastritis, oesophagitis and gall stone dyspepsia[7].

Duodenogastric reflux can occur as a normal physiological event which may assume a pathological role after cholecystectomy due to altered physiology [4]. "Normal reflux" may differ from "abnormal reflux" in frequency, amounts and duration[8].

Loss of reservoir function of gall bladder in cholecystectomised patients causes increase in DGR, due to a constant supply of bile in to the duodenum, even when fasting. Another possibility is that the pyloric mechanism may be impaired in patients after cholecystectomy with subsequent pyloric incompetence[9-11].

There is clinical and experimental evidence that regurgitation of duodenal juice into the stomach is capable of damaging the gastric mucosa and cause symptoms. The composition of bile refluxing in to the stomach is altered after cholecystectomy to a composition that is more damaging to gastric mucosa[12-13].

MATERIAL AND METHODS

The study was conducted in the Departments of Surgery and Nuclear Medicine at SKIMS. The patients selected from Department of General Surgery had not undergone any other prior gastric or hepatobiliary surgery. Patient with choledocholithiasis, surgical obstructive jaundice, cholangitis, known peptic ulcer disease or biliary tract disease other than cholelithiasis were excluded from the study.

Patients were subjected to cholescintigraphy using Tc-99m Mebrofenin before cholecystectomy and between 2-4 months post cholecystectomy. The dynamic serial images were processed on the computer using a pre-defined processing software(Fig. 1). From the computer generated time activity curve reflux in the stomach area was

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calculated(Fig.2).

Pre-surgery 37 patients were included in the study. All the 37 patients underwent preoperative cholescintigraphy and surgery, however, seven patients were lost to follow up and only 30 patients turned up for postoperative evaluation and scanning. Hence the study is based on these 30 patients.

OBSERVATIONS AND RESULTS

Table 1:

S. No.	Patient ID	Age	Sex	Pre-op. Reflux %age	Post-op. Reflux %age
01	NM3	36	F	11	13
02	NM4	38	F	7	9
03	NM5	42	M	13	14
04	NM6	50	F	7	33
05	NM7	45	F	9	16
06	NM8	40	M	10	13
07	NM9	40	F	10	11
08	NM10	50	F	2	37
09	NM11	47	F	21	16
10	NM12	35	F	1	2
11	NM13	52	F	28	33
12	NM14	28	F	6	27
13	NM15	38	M	7	9
14	NM16	28	F	9	12
15	NM17	30	F	22	38
16	NM18	32	F	30	30
17	NM19	35	F	9	12
18	NM20	54	F	6	8
19	NM21	37	F	21	28
20	NM22	55	F	2	34
21	NM23	42	F	2	13
22	NM24	27	F	12	19
23	NM25	37	F	4	5
24	NM26	29	F	10	12
25	NM27	37	F	3	5
26	NM28	45	F	5	7
27	NM29	51	F	15	16
28	NM30	57	F	10	21
29	NM31	47	M	18	18
30	NM32	17	F	9	9

DGR before and after cholecystectomy in the same patients. Insignificant pre-cholecystectomy reflux with significant post-cholecystectomy reflux (n=7) was significant at p value (paired t - test) = < 0.05.

Seven patients showed a significant, more than 14% reflux (DGR) preoperatively. These patients were labelled preoperative refluxes. These patients continued to have a significant reflux postoperatively also.

One patient showed a decrease in DGR from 21 to 16% and 2 patients showed no change in DGR(Table I).

Three of the 7 preoperative refluxers had attended out patients department with recurrent gastritis like picture and bilious vomiting.

The amount and pattern of gastric activity in patients judged to be positive for DGR preoperatively was quite distinct from the amount of back ground activity counted in a comparable left upper quadrant area of the abdominal field in negative studies.

Seven patients with an insignificant reflux preoperatively had an enhanced postoperative reflux (>14%). On statistical analysis this increase was found to be significant at $p<.01$ (paired 't' test). Overall 14 patients had significant DGR postoperatively(Table I).

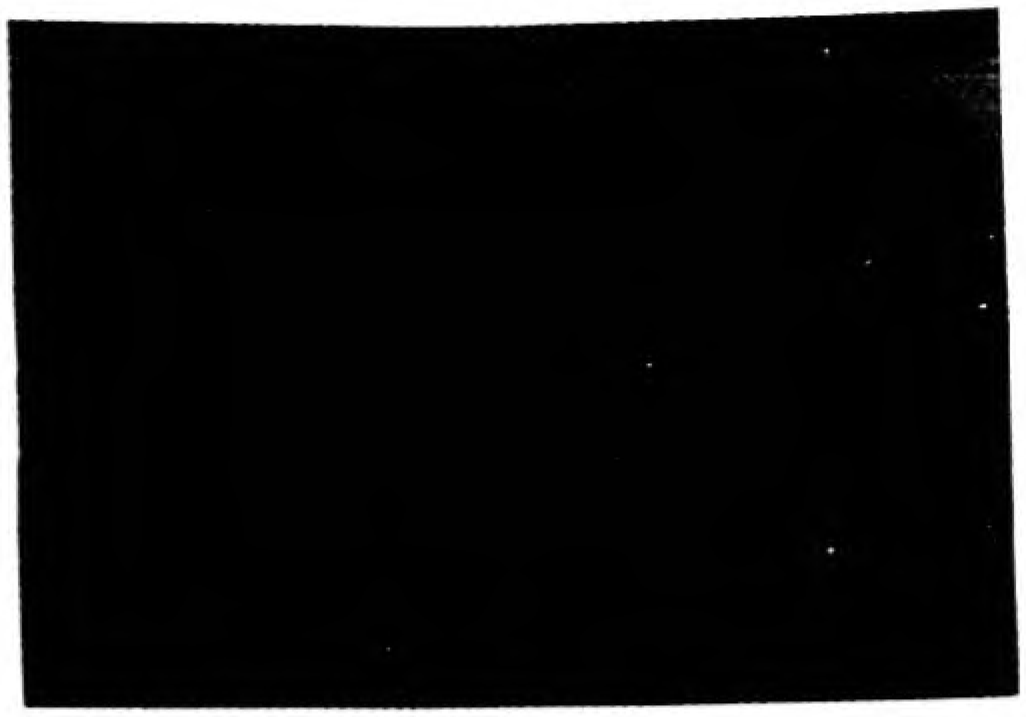

Fig. 1

Fig. 2

Table II

Group	No. of Refluxers	Peak gastric activity Mean (Range)	Time of peak (min) gastric activity mean (range)
Patients of cholelithiasis	7	21.4(15-30)	24.5(00-6.0)
Cholecystectomised patients	14	22.7(16-38)	26.17(00-60)

Based on the above results, it can be established that the number of refluxers i.e., those having significant reflux (DGR) increased post cholecystectomy. 7(23.33%) patients who did not show significant ($p<.01$) preoperative DGR developed post cholecystectomy reflux. Five from this group of 7 patients presented postoperatively with the clinical picture of bile gastritis. Main complaint of these patients was burning epigastric pain and 2 patients developed bilious vomiting denovo.

The mean of individual peak gastric activity and the mean of time of individual peak gastric activity in patients with significant DGR preoperatively and those having significant DGR postoperatively showed more or less matching pattern (Table II).

DISCUSSION

Cholecystectomy though an accepted treatment for patients who are believed to have symptoms due to gall stones has also been under focus as a critical factor in causation of postoperative alkaline gastritis as a result of enhanced DGR.

A significant number of patients continue with the symptoms after cholecystectomy and in some, certain symptoms may even increase in intensity or new symptoms may appear postcholecystectomy. Increased DGR is believed to be a major cause of symptoms after cholecystectomy in patients in whom other probable causes have been ruled out. Recent studies have pointed towards enhanced DGR reflux after cholecystectomy[10]. The bile flow into the duodenum is unregulated and more continuous after cholecystectomy.

A syndrome has emerged in which the constant features are upper abdominal pain usually burning in nature and aggravated by eating and gastroscopic evidence of superficial gastritis confirmed by biopsy. Associated features are nausea, bilious vomiting etc.

Gastric mucosal injury with resultant symptoms could be attributed to any one or a combination of the following possibilities[4,9,14].

1. Enhanced DG reflux due to loss of gall bladder reservoir function.
2. Enhanced DG reflux due to pyloric incompetence after cholecystectomy
3. Postoperative change in the composition of bile with more of unconjugated salts more injurious to gastric mucosa.

From the preoperative symptomatology, nausea, vomiting and abdominal pain were observed to be specific to gall stone disease and have a high probability of being cured by surgery whereas symptoms like belching, heart burn, sour eructation and intolerance to fatty food are not. These findings were also observed in other studies[15].

Dyspepsia was not considered specific to gall stones being equally common in patients with or without gall stones, but patients with gall stones and dyspepsia continue to undergo cholecystectomy.

The postcholecystectomy symptoms of patients who had significant DGR did not differ much from patients who did not have a significant DGR. One major difference however, we noted in our study was bilious vomiting present in 6 patients postoperatively. This included 4 patients who had bilious vomiting preoperatively and 2 patients developed it de novo.

Incidence of significant DGR in our study increased from 23.3 percent preoperatively to 46.6 percent postoperatively. Patient symptoms could be attributed to DGR in four patients preoperatively and in six patients postoperatively. DGR has been implicated in causations of gastritis in other studies also[10]. Although postcholecystectomy symptoms can be attributed to DGR in a limited number of patients. It is possible that over a longer period of follow up the number of such patients may increase.

CONCLUSION AND RECOMMENDATIONS

Evaluating patients with gall stones and dyspepsia also for DGR in addition to other investigations before surgery might hold some merit. The refluxers could then have a different management from non-refluxers although surgery for cholelithiasis remains the corner stone of any treatment plan.

Patients with significant DGR may need further evaluation and should be made aware of the possibility of persistent symptoms or appearance of new symptoms after cholecystectomy.

REFERENCES

1. Venu RP and Geenen JE. Postcholecystectomy syndrome. Textbook of Gastroenterology, Second edition. Edited by Tadatska Yamada. JB Lippincott Company Philadelphia 1995.
2. Cheadle WG, Patti V, Mackie CR and Cuschieri A. Effect of gall bladder function on duodenogastric reflux. Gut 1984;25: 1138.
3. Brown TH, Walton G, Cheadle WG, Larson GM. The alkaline shift in gastric pH after cholecystectomy. Am J Surg 1989;157.
4. Svensson JO, Gelin J and Svanvik J. Gall stones, cholecystectomy and duodenogastric reflux of bile acid. Scand J Gastroenterol 1986:21:181-7.
5. Muller-Lissner SA, Sonnenberg A, Muller-Duysing W, Will N, Heinzel F and Blum AL. Quantitative measurement of duodenogastric reflux in man. Sand J Gastroent 1981;16(suppl 67):43-6.
6. Campbell FC, Kennedy N, Sutton D, Paton G and Cuschieri A. Gall bladder function and fasting enterogastric bile reflux. Br J Surg 1987; 74(6).
7. Harding LK, Sorgi M, Wolverson RL, Mosimann F, Sherwin S, Donovan IA, Alexander -Williams J. The pharmacokinetics of 99m Tc-HIDA in man and its relationship to intragastric bile acids. Scand J Gastroenterol 1984;19(suppl 92):27-9.
8. Mackie C, Hulks G and Cuscheiri A. Enterogastric reflux and gastric clearance of refluxate in normal subjects and in patients with or without bile vomiting following peptic ulcer surgery. Ann Surg 1986;p537-42.
9. Warshaw AL. Bile gastritis without prior gastric surgery: Contributing role of cholecystectomy. The American Journal of Surgery 1979;137:527-31.
10. Brough WA, Taylor TV and Torrance HB. The effect of cholecystectomy on duodenogastric reflux in dogs and humans. Scand J Gastroenterol 1984;19(suppl 92): 242-4.
11. Muller-Lissner SA, Fimmel CJ, Sonnenberg A, Will N, Maller Daysing W, Heinzel F, Muller R and Blum AL. Novel approach to quantitate duodenogastric reflux in healthy volunteers and in patients with type -1 gastric ulcer. Gut 1983;24:510-8.
12. Ritchie WP Jr. Postoperative alkaline gastritis: A prospective clinical study of etiology and treatment. Scand J Gastroent 1981;16(supp 67):233-5.
13. Ritchie WP. Alkaline reflux gastritis: an objective assessment of its diagnosis and treatment. Ann Surg 1980; 92:288-98.
14. Heading RC. Dudenogastric reflux. Gut 1983;24:507-9.
15. Stroker DL, Williams JG, Macleod MA, Colin-Jones DG. The evaluation of a gastric bile probe. Nucl Med Commn 1990;1:777-90.

RETROPERITONEAL LAPAROSCOPIC NEPHRECTOMY FOR BENIGN RENAL DISEASE

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Abstract: *Lot of advances have been made in the field of minimal access surgery. Procedures like Laparoscopic Cholecystectomy, Appendectomy, repair of Inguinal hernias, correction of GERD, etc. have been performed successfully. However, removal of large and solid organs using laparoscopic apporach present obvious difficulties to the surgeon. Laparoscopic Nephrectomy can be performed via the transperitoneal as well as via the retroperitoneal route for both benign as well as malignant conditions of the kidney. Here we present our experience of Laparoscopic Nephrectomy for benign conditions of the kidney using the retroperitoneal approach. Eleven patients with benign renal disease were operated between March and September 2002, at SMHS Hospital, Srinagar. All patients were operated through the retroperitoneal approach using only three ports (10mm, 5mm and 5mm). In ten patients the procedure was performed entirely laparoscopically. One patient required conversion to open nephrectomy due to bleeding from the renal vein. This patient had dense adhesions due to tuberculosis of genitourinary system. Operative time ranged from 45 minutes to 1 hour and 25 minutes. Median post operative stay was 3.7 days.*

Retroperitoneal Laparoscopic Nephrectomy appears to have a defnite role in the removal of kidneys for benign disease. Based on our experience this technique results in very few complications. The technique utilizes only three port sites and sometime an additional port. The peritoneum is not breached, reduced post operative pain, early mobilization, early discharge from the hospital and early resumption of normal activities are all advantages over the conventional open procedure.

Key words: *Nephrectomy, Laparoscopy, Retroperitoneal, Benign disease.*

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INTRODUCTION

Major advances have been made in the field of minimal access surgery during the last decade.Uptill two decades ago laparoscopy was used primarily as a diagnostic tool. With the advances in the instrumentation and techniques of laparoscopy and the acceptance of laparoscopic cholecystectomy as the procedure of choice, many other intra abdominal procedures are being performed laparoscopically. Although laparoscopic nephrectomies have been performed for both benign[1] as well as malignant[2] disease process by both transperitoneal and retroperitoneal routes with advantages over the conventional open procedure[3], here we report our experience of retroperitoneal laparoscopic nephrectomy for benign disease.

PATIENTS AND METHODS:-

From March 2002 to September 2002, 11 patients underwent retroperitoneal laparoscopic nephrectomy using standard laparoscopic surgical equipment at SMHS hospital,Srinagar,Kashmir. Patient details and preoperative investigations are given in Table1.

Patients who had undergone any renal surgery on the same side were excluded from the study. All patients were admitted to hospital one day prior to surgery.

OPERATIVE TECHNIQUE:

Following induction of general anesthesia, the patient was placed in the standard kidney position and strapped to the operating table. A 10 to 14 mm incision was made just 1 cm below the angle of the12th rib. All muscle layers where split using a hemostat through this incision following which the index finger of the dominant hand was used to feel the retroperitoneal fat and make space for the balloon trocar.The balloon trocar was then inserted through the incision and inflated upto 800 to 1000 cc.This facilitated creation of a retroperitoneal space. During inflation a 10 mm telescope was then inserted through the balloon trocar and the retroperitoneum viewed through the balloon. The balloon was deflated and removed and a standard 10 mm trocar was inserted for the telescope. The two working ports were inserted under direct vision. The first 5 mm working port was inserted 2 cms above the anterior

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TABLE 1:

PATIENT	SIDE OPERATED	SEX	AGE	SYMPTOM	DIAGNOSIS	PREOPERATIVE INVESTIGATIONS.
1.	RIGHT	M	45	Right Flank pain	Right PUJ obstruction	Ultrasound, IVP, DTPA Scan – 12 % function
2	RIGHT	M	48	Right Flank pain, chills, Rigors, fever	Right hydro nephrosis and Hydroureter sec. to calculi	Ultrasound IVP,DTPA scan- 14% function PCN
3	LEFT	M	37	Left Flank pain Hematuria	Gross hydro nephrosis impacted proximal ureteric calculus	Ultrasound IVP,DTPA scan- 10% function
4	RIGHT	F	50	Right Flank pain, chills, fever	GUTB	Ultrasound IVP,ELISA for TB,urine for AFB C/S
5	LEFT	F	29	Left Flank pain	Gross hydro nephrosis conginital PUJ obstruction	USG, IVP,DTPA scan 8%function
6	RIGHT	M	40	Right Flank pain, chills, Rigors, fever	Chronic pyelonephritis Gross hydro nephrosis	USG, IVP PCN
7	RIGHT	F	42	Right Flank	Enlarging stag horn calculus with Gross hydro nephrosis	USG,IVP DTPA –14% function
8.	RIGHT	M	38	Right Flank pain with fever	Right PUJ Obstruction	USG,IVP,PCN DTPA scan 10% function
9.	RIGHT	F	54	Right Flank pain with fever and dysuria	Chronic Pyelonephritis with Gross Hydronephrosis	USG, IVP, PCN
10.	LEFT	F	42	Left Flank pain	Stag Horn calculi with Gross Hydronephrosis	USG, IVP, DTPA scan- 8.4% function
11.	RIGHT	M	45	Right Flank pain	Right PUJ Obstruction	USG, IVP, PCN

superior iliac spine in the anterior axillary line. The second 5mm/10mm working port was placed at the renal angle. All the three ports were in line with each other so that in case of conversion all would lie in the line of incision. After the placement of the ports the ureter and hilum were identified. We did not find placement of the ureter catheter necessary in any of our patients[2]. Since all of our procedures used the retroperitoneal route, the administration of bowel preparation and use of nasogastric tube prior to operation was not thought necessary[2]. Using blunt and sharp dissection the vessels at the hilum were defined.Hilum dissection was done from the posterior approach.Following this intracorporeal ligature of the vessels or application of ligaclips was done depending on the size of the vessels. Dissection of perinephric fat and adhesions all around the kidney was done except at the top from where the kidney remained suspended. Ureter was identified and freed from the posterior abdominal wall.The ureter was ligated with clips or a ligature was tied intracorporally.Lastly the attachment at the top from which the kidney remained suspended was taken down. The freed kidney when infected was then placed in an endobag whose neck was brought out of the skin through 10 mm trocar site. During this time a 5 mm telescope was insertedthrough port no 2 to view the procedure. The kidney was then extracted intact by enlarging the trocar incision by 2 to 4 cms. After careful inspection of the operative site, all trocars were removed under direct vision. A Redivac vacuum drain was then placed through the 5 mm port site. The other port sites were closed with 2-0 vicryl suture. The skin was closed with 2-0 interrupted silk sutures.

DISCUSSION:-

Minimally invasive techniques have replaced open surgery not only for the general surgeon but for urological conditions as well . Although laparoscopic cholecystectomy[4], appendectomy[5], repair of ingunial hernia[6], correction of GERD have all been performed with great success, the removal of large organs like kidneys and spleen present obvious difficulties. Our study was aimed to determine whether laparoscopic nephrectomy would be of benefit to patients when used in selected cases.

In the present study, 11 patients with benign renal disease where taken up for the procedure.Patients who had previously undergone any renal surgery on the same side were excluded from the study. One patient required conversion to open procedure due to inability to control bleeding from the renal vein. This patient had dense adhesions due to tuberculosis of the kidney and subsequent difficult dissection. All other 10 patients were operated

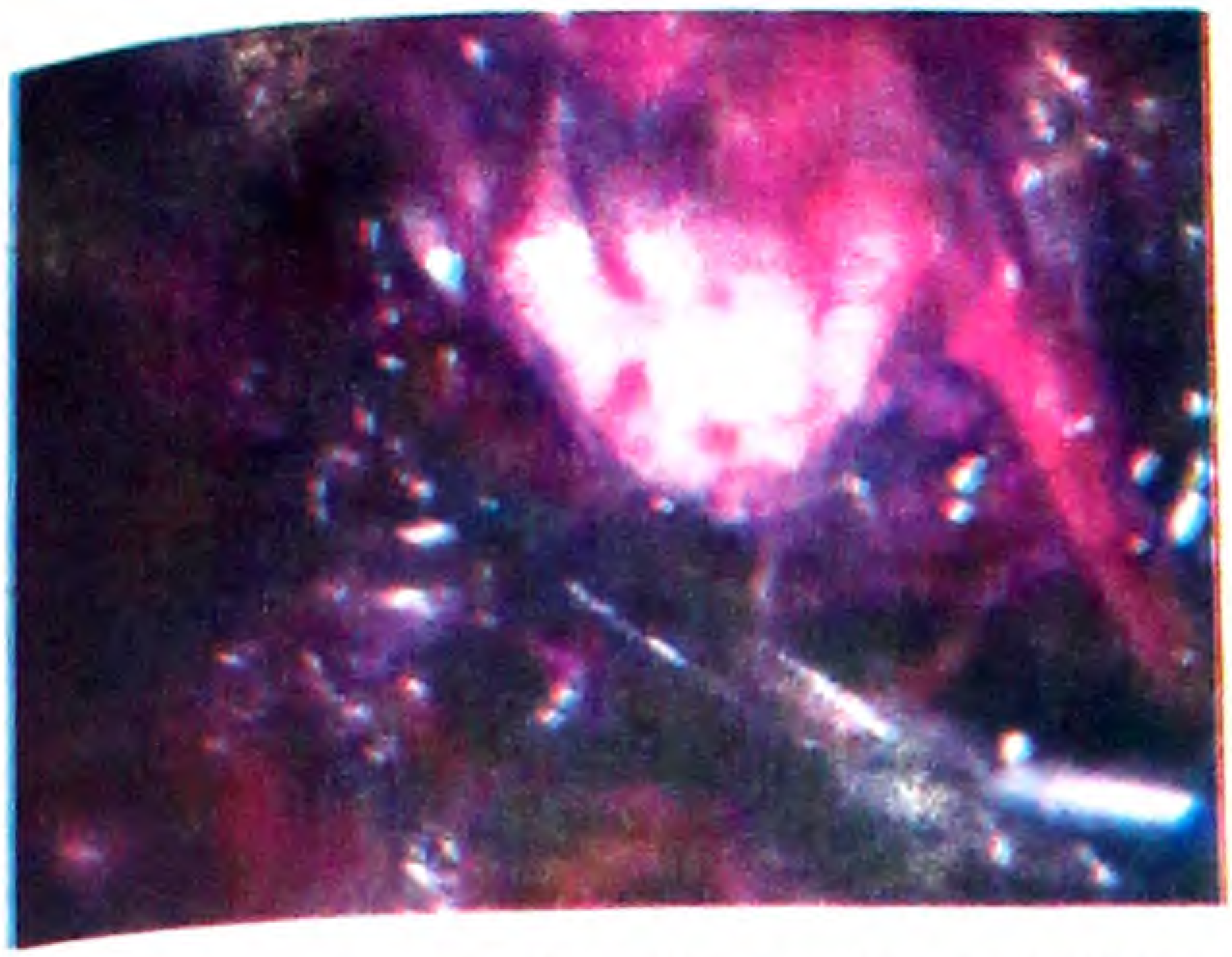

Fig. 1: Intracorporeal knotting of Renal Pedicle

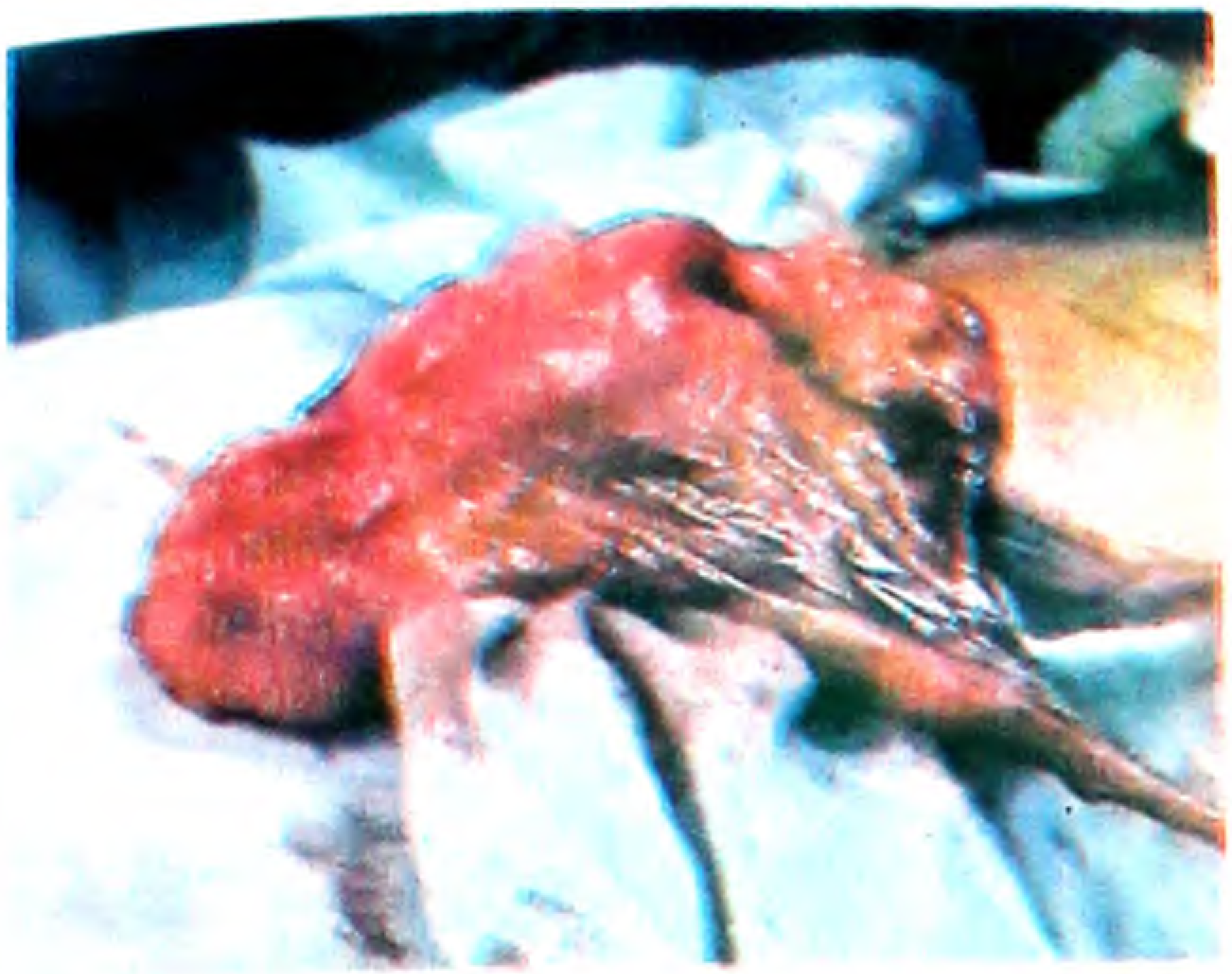

Fig. 2: Intact Nephrectomy Specimen after retrival from 1.5cm fort.

laproscopicaly through the retroperitoneal route. We used only 3 port sites successfully in 8 of our patients and 4 port sites in 2 patients. We found that by approaching the hilum first,the need for additional retractors and ports is obviated. The need for mechanical and antibacterial bowel preparation and NG tube placement was omitted by using the retroperitoneal approach. Mechanical and antibacterial bowel cleansing increase required hospitalizations and costs and they are unpleasant to patients. Also placement of the ureteric catheter was not necessary as in our experience this did not affect our identification and dissection of the ureter. Both these steps add to the cost of the procedure as well as to the operating time. Since all of the procedures were performed for benign conditions of the kidney and the renal vessels were small,they could be controlled early by intracorporeal ligature or ligaclips thus obviating the use of expensive GI stapler for control of hilar vessels.

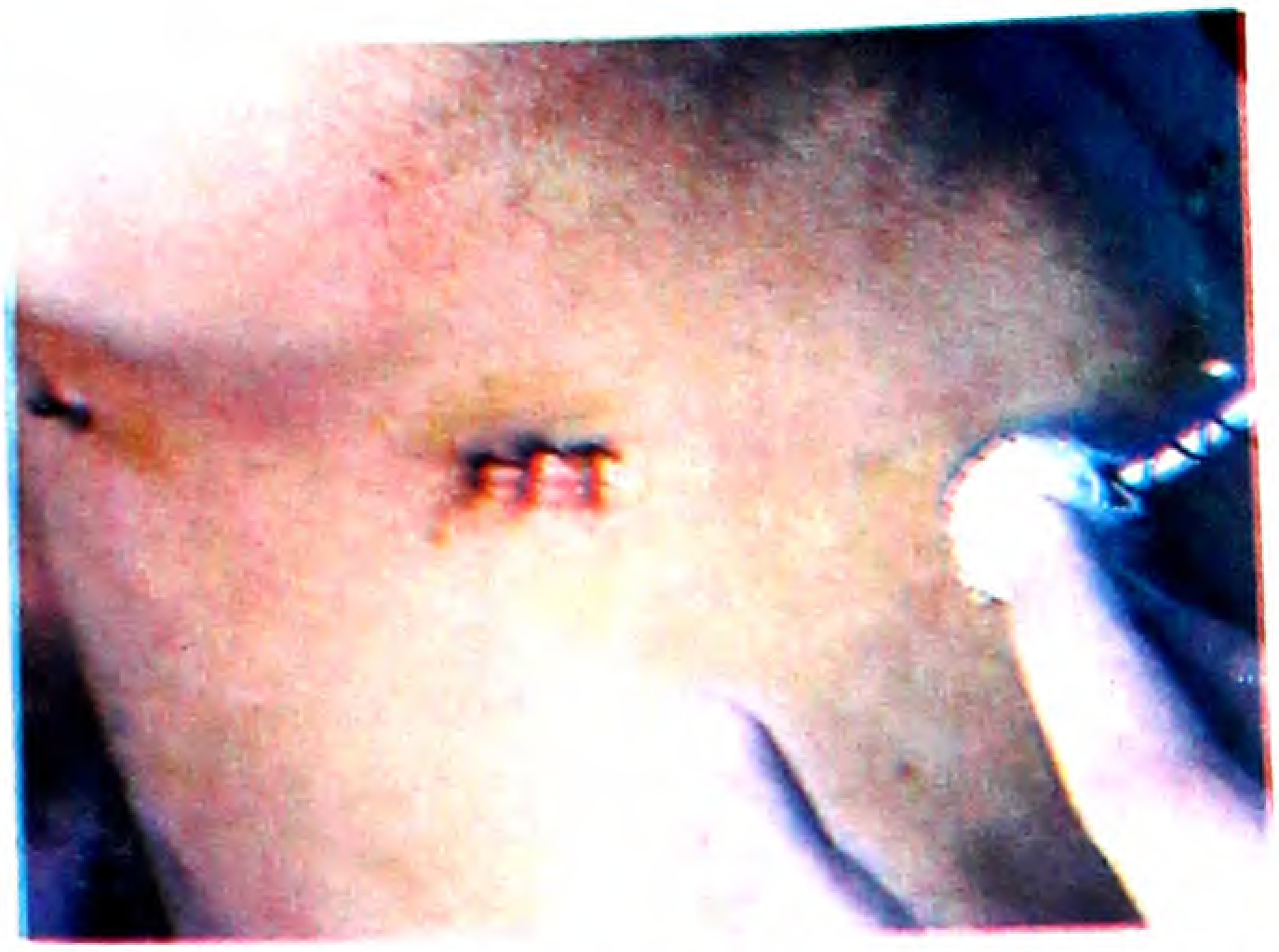

Fig. 3: External View of Port Sites after completion of Procedure.

RESULTS:

Operative and postoperative details are in Table II. Operating time ranged from 45min to 1hour 25 minutes. Median postoperative hospitilization was 3.7 days (range 3-5days).

During the procedure in 2 patients the peritoneum was inadvertently opened which decreased the operating space during the procedure. It was sutured back with 2-0 catgut using intracarporal suturing technique at the end of the procedure. One patient required conversion to open nephrectomy due to bleeding from right renal vein. This patient had dense adhesions from renal tuberculosis. One patient with chronic pyelonephritis developed superficial wound infection of the incision used to deliver the kidney. 3 patients developed minor surgical emphysema around the 10mm port site which disappeared 12 hours after the procedure without any intervention. Because of non availability of a tissue morcelator at our center we were not able to avoid the need for extention of the incision at the 10 mm trocar site for extraction of the kidney. 3 patients needed enlargement to only 2cm incision at the 10mm trocar site due to loss of pacenchyma of the kidney by the disease process with resultant small size of the kidneys. All other patients needed enlargment of the 10 mm trocar site to 3 - 4cm for kidney retrival.

Further, the use of intracorporal suturing of the vascular pedicle also decreased the total cost of the procedure,

TABLE II:-

PATIENT	VASCULAR PEDICLE	OPERATIVE TIME (HRS)	DISCHARGE RETRIVAL	KIDNEY	COMPLICATIONS
1	Intracorporal Suturing	1 hour and 10 min	Day 3	4cm incision at 10mm trocar site	Minor surgical emphysema at 10mm trocar site
2	Intracorporal suturing	1 hour and 25 min	Day 3	4cm incision at 10mm trocar site	Inadvertent opening of the peritoneum intraoperatively
3	Ligaclips	55min	Day 4	2cm incision at 10 mmtrocar site	None
4	Conversion to open nephrectomy				
5	Intracorporal suturing	1 hour and 20 min	Day 4	4cm incision at 10 mm trocar site	None
6	Ligaclips	45 min	Day 5	4cm incision at 10 mm trocar site	Superficial wound infection at kidney retrival site Surgical emphysema at 10- mm trocar site
7	Intra corporal suturing	1hour and 5 min	Day 3	2cm incision at 10 mm trocar site	None
8	Intra corporal suturing	1hour and 15 minutes	Day 3	3cm incision at 10 mm trocar site	None
9	Intra corporal suturing	1 hour and 20 minutes	Day 5	4cm incision at 10 mm trocar site	Superficial wound infection at kidney retrival site.
10	Intra corporal suturing	1 hour and 25 minutes	Day 4	4cm incision at 10 mm trocar site	Inadvertent opening of the peritoneum intraoperatively, surgical emphysema at 10mm trocar site
11	Ligaclips	50 min	Day 3	2 com incision at 10 mm trocar site	None.

although this minimally increased the operating time of the procedure.

Although previous experiences with laproscopic nephrectomies of different authors had shown an increase in the operating time[1], we did not find a significant difference between the operating time of laparoscopic and open nephrectomies. Also the small incision causes less post operative pain,shorter post operative stays and earlier resumption of normal activity. In all cases in our series the mean hospital stay (3.7 days) was less than that reported in a series of healthy patients undergoing live donor nephrectomies[7] and[8] . Also 4 out of the 10 patients in our study were obese otherwise needing large incisions to remove their kidneys by open surgery. By avoiding this , these patients were mobilized earlier with no thromboembolic or pulmonary complications.

To conclude from our limited experience laparoscopic retroperitoneal nephrectomy has a definite role in the removal of kidneys with benign diseases in selected patients. This technique has a definite advantage offering decreased post operative pain ,early ambulation and decreased post operative hospital stay, especially in the obese patient. With further experience the operating time is bound to decrease further. Also the many complications like Incisional hernias, pulmonary and thromboembolic complications are done away with. Laparoscopic nephrectomy is also cost effective compared with open nephrectomy if short operating times and brief length of stays are achieved.

References:

1. DL Nicol,DC Winkle,LK Nathanson,BM Smithers.Laparoscopic nephrectomy for benign renal disease.British Journal Of Urology,1994,73;237-241.
2. Louis R.Kavoussi, Kurt Kerbl, Carl C. et al.Laparoscopic nephrectomy for renal neoplasms.Urology,Nov 1993,vol42,Number 5.
3. Lotan Y, Gettman MT, Roehrborn cg, Pearle MS, Cadeddu JA.Cost comparison of Laparoscopic Nephrectomy and Open Nephrectomy: analysis of individual parameters.Urology;2002 June;59(6):821-5.
4. Larson GM,Vitale GC,Casey J et al.Multipractice analysis of Laparoscopic Cholecystectomy in 1983 patients.Am J Surg 1992;163:221-6.
5. Gangal HT, Gangal MH. Laparoscopic appendectomy. Endoscopy 1987;19:127-9.
6. Salerno GM, Fitzgibbons RJ, Filipi CJ. Laparoscopic Inguinal hernia repair.Surg Laparoscopy,St Louis:Quality Medical Publishing: 281-93.
7. Weinstein SH, Navarre Jr RJ, Loening SA et al. Experience with live donor nephrectomy.J Urol 1980;124:321-3.
8. DeMarco T, Admin M, Harty JL. Living donor nephrectomy:factors influencing morbidity.J Urol 1982;127:1082-3.

EXTERNAL FIXATION OF INTERTROCHANTERIC FRACTURES OF THE FEMUR.

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Abstract: External fixation was used in the treatment of 12 intertrochanteric fractures of the femur from March 2002 to January 2003, at Bone & Joint surgery hospital Srinagar. Good fixation and early ambulation was achieved in all cases. Blood loss was negligible. Pin tract infection occurred in one case and pin loosening in one case. The average time of union was 16 weeks.

The surgical technique is simple, inexpensive, is a quick procedure and causes minimal surgical trauma. These features are particularly relevant where resources are limited.

Key Words: *intertrochanteric fractures, external fixator, internal fixation, skeletal traction.*

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Introduction:

The aim of treatment of intertrochanteric fractures is to achieve a union in good position with least discomfort to the patient[1-4]. In a developing country like ours, some patients with intertrochanteric fractures are not suitable for any treatment by conservative means or conventional internal fixation. External fixation after closed reduction seemed to fulfil all requirements of treatment listed above. It may not be a very superior method to Richards fixation[4-8], but definitely a superior method to boot and bar method of treatment.

Materials and Methods:

12 cases of intertrochanteric fractures of femur were treated, with External Fixator from Mar 2002 to Jan 2003, averaging a mean follow up of 30 weeks. The categories of patients treated are given in Table 1.

Table 1:

Description.	Number.
Alzheimers Disease.	1.
Diabetics.	3.
Cardiac Disease.	2.
Geriatric.	5.
Patient with bed sore.	1.

Sex Distribution:

Table 2:

Male.	8.
Female.	4

Patients were investigated pre-operatively and only those considered to be poor risks for conservative and conventional surgery(ORIF), were included in this study. We did not classify the fractures into stable and unstable groups, as the decision to operate was based upon the general condition of the patient.

Technique:

Patients were given Injection Pentazocin intravenously, and Injection Diazepam was also given in some cases. Local anesthetic in the form of 2% Xylocaine was infiltrated at the pin tract sites. Patients were taken on the fracture table and after reducing the fracture under fluoroscopic control, a guide wire at an appropriate angle was passed to the neck and head of femur. Two 5mm Schanz srews were passed through the base of the greater Trochanter across the fracture site to the head and neck of femur. Three 5mm Schanz screws were inserted at right angles into the middle half of of the femoral shaft. Clamps were tightened and final position checked under the C-arm.

Pin tract cleaning was done daily with hydrogen peroxide, saline and betadine. Intravenous antibiotics and analgesics were given for three days and followed by oral antibiotics for three days.

Static Quadriceps excersices were started immediately with active Hip, Knee and Ankle mobilization in the bed. After 1 week the patients were made to sit up in the bed. At 3 weeks, patients were allowed to hang their feet by the side of the bed. Non weight bearing ambulation was started at 3 weeks. The patients graduated to partial weight bearing with crutches at 6 weeks. This was followed by full weight bearing at 10 weeks.

Serial x-rays were done every three weekly and after

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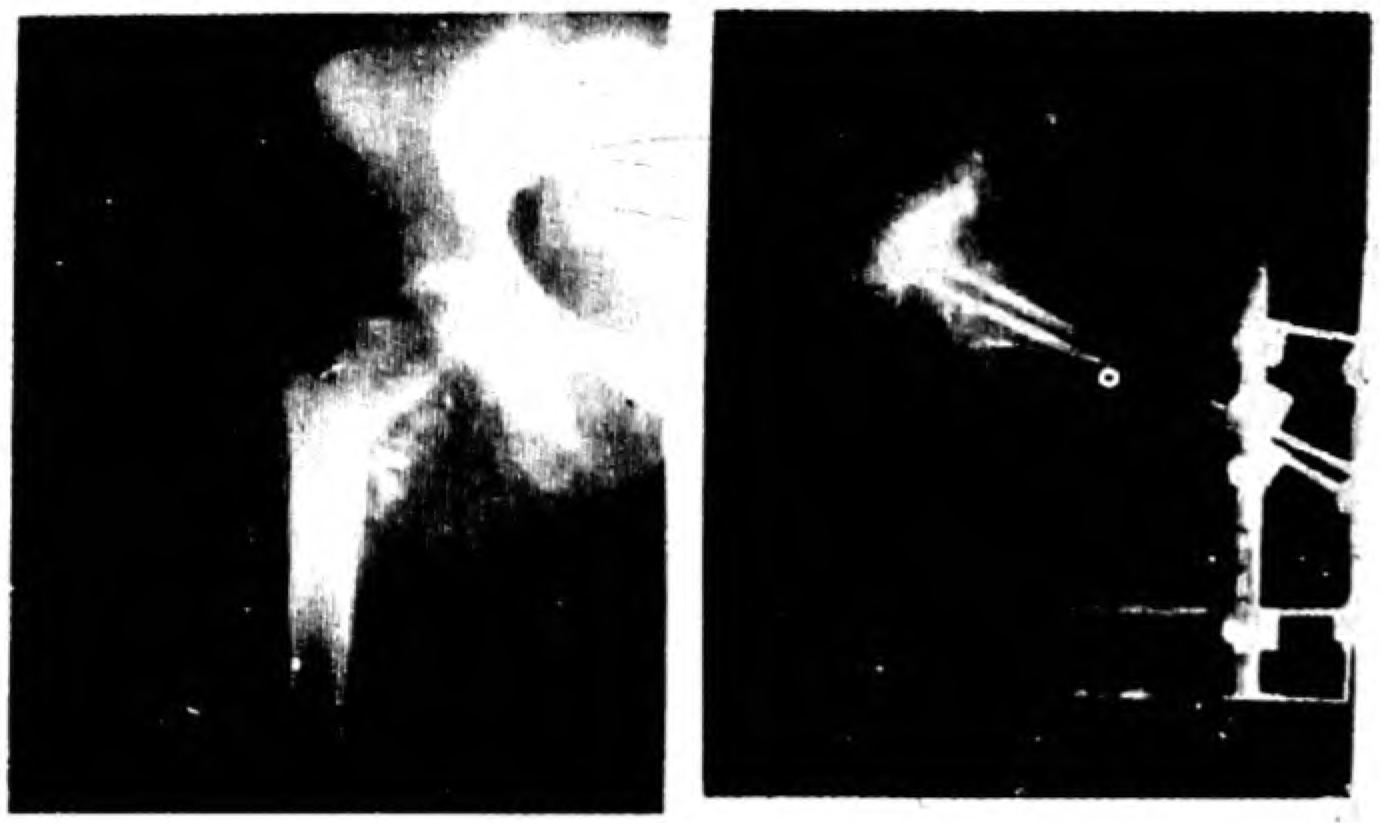
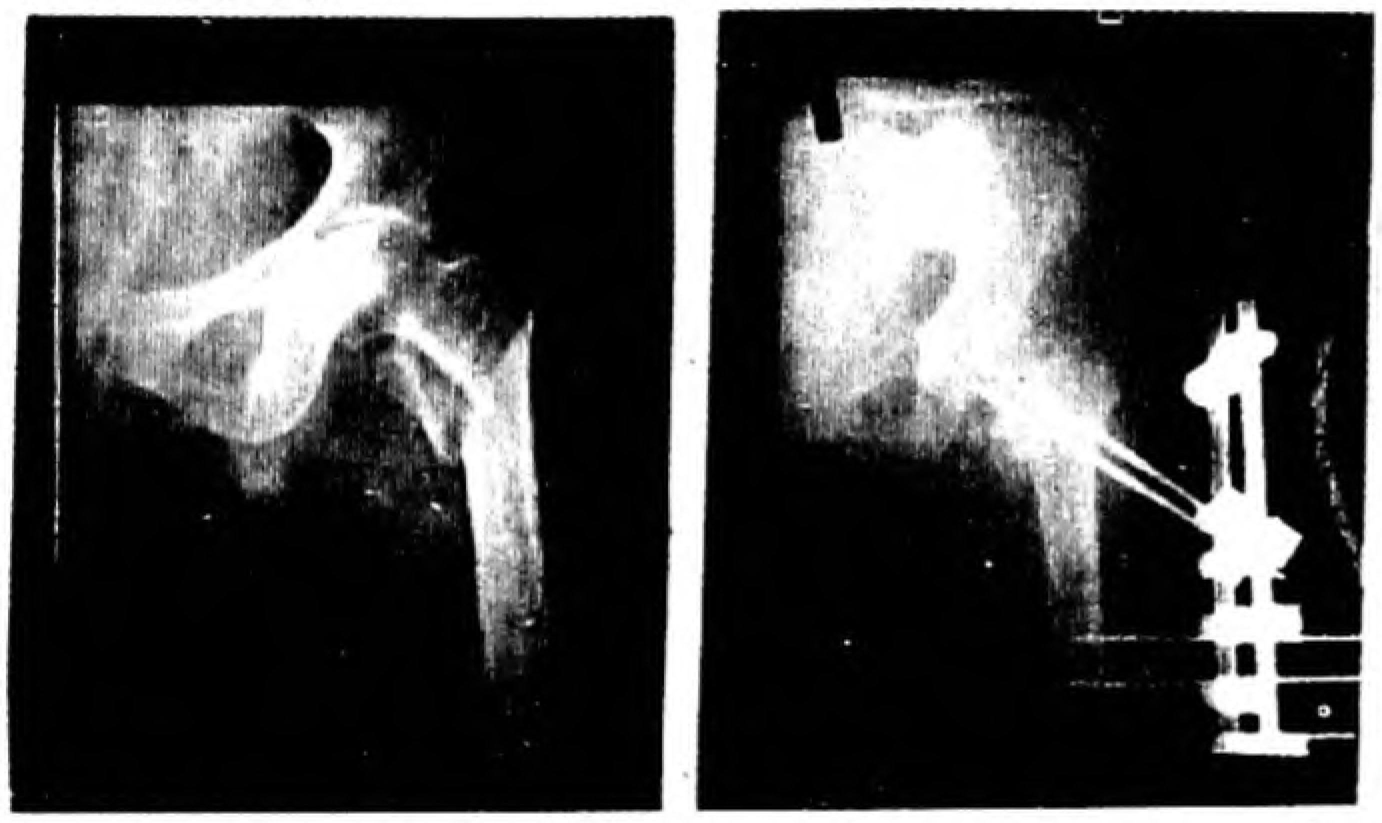


Pre-op(ap). Post-op(ap).

x-Rays: (Fig 1.)



Pre-op(ap). Post-op(ap).

Fig.2

clinical and radiological signs of fracture union,the Fixator assembly was loosened and stress views were taken and depending on the above,the external fixator assembly was removed.

Results:

From March 2002 to January 2003, 12 cases of intertrochanteric fractures were treated by external fixator,with an average follow up of 30 weeks(16-44 weeks). There were 8 males and 4 females. The age of the patients varied from 56 years to 76 years(avg. 66yrs).The average time of union was 16 weeks.

Table 3:

Complications.	*Number.*
Pin tract infection.	1.
Pin lossening.	1.

Pin tract infection occurred in one patient which was treated by aggressive antibiotic therapy and frequent dressings. Pin loosening occurred in one patient which needed a revision of schanz screw.

In 10 patients there was a shortening of less than 1 cm,and in two patients a shortening of more than 1 cm,this was probably due to fixation in varus and collapse of the neck shaft angle on weight bearing. Intial Knee stiffness was noted in every case.The range of knee motion was 20-30 degrees initially,which gradually improved over a period of time by physiotherapy.

Discussion:

The frequency of intertrochanteric fractures in our population is high,accounting for almost 1 case in every 8 orthopaedic emergency admissions.The use of external fixator was preferred in all the cases in this study due to their lack of fitness for undergoing major surgical trauma,under any type of anesthesia as well as prolonged recumbency.The choice of patients for this procedure is reflected in Table 1.Acheiving union is not a problem since intertrochanteric fractures are through highly vascular cancellous bone[1], the problem is to maintain or reconstitute the neck shaft angle. The best that a surgeon can achieve is to secure this position while allowing the patient to enjoy early ambulation(1,3). The most effective conservative method is Skeletal traction.The results of this method are good provided the patients can survive the hazards of prolonged recumbency. In our hospital with a bed occupancy rate of 120%,keeping the patients on traction for 8-12 weeks would be difficult. A major advantage of external fixation is that the average stay in the hospital is less than 2 weeks.

Although external fixator has been in vogue for quite a long time, however there have been very few previous reports for its use in the intertrochanteric fractures.The reasons probably might include;

◆ the fear that external fixator pins transfixing the fracture could cause infection at that site,our experience has shown that this does not occur.

◆ the external fixator cannot provide rigid immobilization,our method provided a semi rigid fixation.However this could be an advantage in unstable intertrochanteric fractures.The rate of union in our series implies that external fixation provides adequate immobilization.

The knee stiffness is probably the result of schanz screws transfixing the vastus lateralis muscle,but it was temporary in all our cases as the knee motion was normal after few weeks of removal of fixator assembly.

Conclusion:

External fixator for intertrochanteric fractures offers advantage of preserving the fracture hematoma,minimal surgical trauma,negligible blood loss,early ambulation,a shorter hospital stay,and removal as an outpatient procedure.

It is simple to perform,inexpensive and yet provides good results.

Refrences:

1. Essar MP;Kassab JY;Jones DHA: Trochanteric fracture of femur. JBJS(Br) 1986;68-B:557-60.
2. Winter WG: Non operative treatment of proximal femoral fractures in the demented,non ambulatory patients. Clin. Orthop 1987;218;97.
3. Dhal A;Varghese M;Bhasin VB: External fixation of intertrochanteric fractures of femur.JBJS 73-B;No:6,Nov1991.
4. Khanna N;Jagaisai J: Bombay hospital Journal(source Internet).
5. James ET;Hunter GA: The treatment of intertrochanteric fractures:a review article.Injury 1983;14:421-31.
6. Jenson JS;Sonne-Holm S;Tondevold E: Unstable trochanteric fractures:a comparative analysis of four methods of internal fixation. Acta orthop Scand 1980;51:949-62.
7. Sernbo I;Johnell O;Gentz CF;Nilson JA: Unstable intertrochanteric fractures of hip,treatment with Ender Pins compared with compression hip screw.JBJS(Am)1988;70-A:1297-303.
8. Mohanty SP;Chaco V: The comparative analysis of operative and non operative management of trochanteric fractures. Indian Orth.Journal.Jan1984;18:1.

INTRAOSSEOUS GANGLION: AN EXTENSION OF GANGLION OF SOFT TISSUES OR VICE VERSA.

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Abstract: *Intraosseous ganglion have been reported in different bones but there are only few published reports demonstrating their communication with soft tissue garglion. 3 among 4 cases reported here have radiographic & operation documented soft tissue extention of intraosseous ganglia.*

Key Words: *Intraosseous - ganglion*

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Introduction

A ganglion is a mucous filled cyst, with a fibrous wall, and is not a true neoplasm. It is typically connected to an underlying joint capsule or tendon sheath by a stalk. They occur most commonly in the hand and are found more often in women. The most common sites are the dorsal carpus, generally arising from the scapholunate ligament, or at the palmar wrist, with origin from scaphotrapezoid and / or trapeziometacarpal joints. They often occur spontaneously as well as after trauma and in patients with OA.

Intraosseous ganglion is classified by WHO as a benign cystic and often multiloculated lesion made up of fibrous tissue, with extensive mucoid changes, located in the subchondral bone adjacent to a joint. Radiologically it appears as a well-defined osteolytic lesion with a surrounding area of sclerosis. It has been described as a synovial cyst, but it lacks a synovial lining. Ganglion cysts of bone occur typically in middle aged men at the ends of long bones, particularly the distal tibia. They are thought to be intraosseous extensions of ganglia of local soft tissues. They have very rarely been reported in proximal radius[1], lunate[2,3], ankle[4] . We report four cases of this rare entity with three of them having clinical, radiological and operation documented extension of local soft tissue ganglia.

MATERIAL AND METHODS

Intraosseous ganglia are believed to be soft tissue extensions of ganglia of local soft tissues but there are only few published reports to confirm this fact. A series of 4 cases of this rare entity is described with 3 among them having clear extension in the local soft tissues.

CASE 1:

A 34 year adult male reported with dorsal wrist ganglion of 4 months duration on right side. He had dull aching of 9 months duration in the same wrist. Clinical examination revealed a tense cystic swelling over mid dorsum of the wrist. X-ray showed a lytic lesion of lunate with overlying round soft tissue shadow along dorsal aspect. CT confirmed the lytic lesion in lunate with defect in the dorsal cortex of lunate through which the contents are communicating with soft tissue ganglion (Fig 1). Operative findings confirmed the findings. Excision of the soft tissue ganglion was done along with curettage of the bony lesion. There was no recurrence of the lesion after one year of follow up.

CASE 2:

A 30-year-old male presented with deep tense cystic swelling in right cubital fossa of 6 months duration. X-ray showed a lytic lesion of neck of the radius opening along anterior aspect of the radius and showing a soft tissue shadow at same level anteriorly (Fig 2). This was confirmed per-operatively and soft tissue ganglion was excised along with curettage of the bony defect. There was no recurrence of the lesion after 4 years of follow up.

CASE 3:

A 25-year-old female complained of pain in lower tibia. X-ray showed a small 1-sq. cm. osteosclerotic lesion just at the base of medial malleolus of right lower limb. CT scan confirmed a sclerotic focus with a radiolucent centre (Fig 3). Excision biopsy of the lesion confirmed the diagnosis. There was no recurrence after 3 years of follow up.

CASE 4:

A 60-year-old lady reported with pain and swelling along anterior aspect of right ankle of 6 months duration. X-ray

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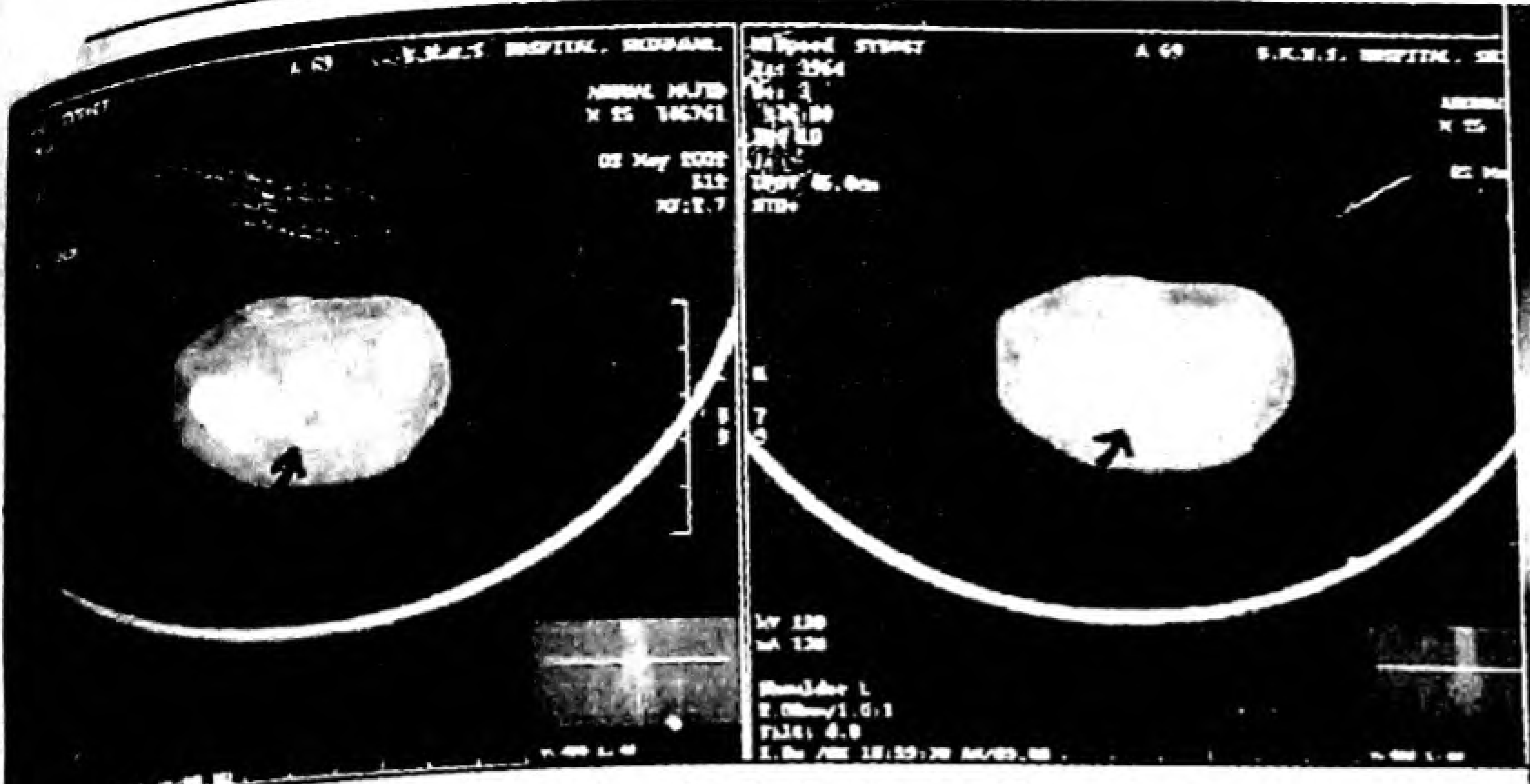

Fig 1: CT scan of wrist showing lytic lesion of lunate. It was communicating with dorsal wrist ganglion.

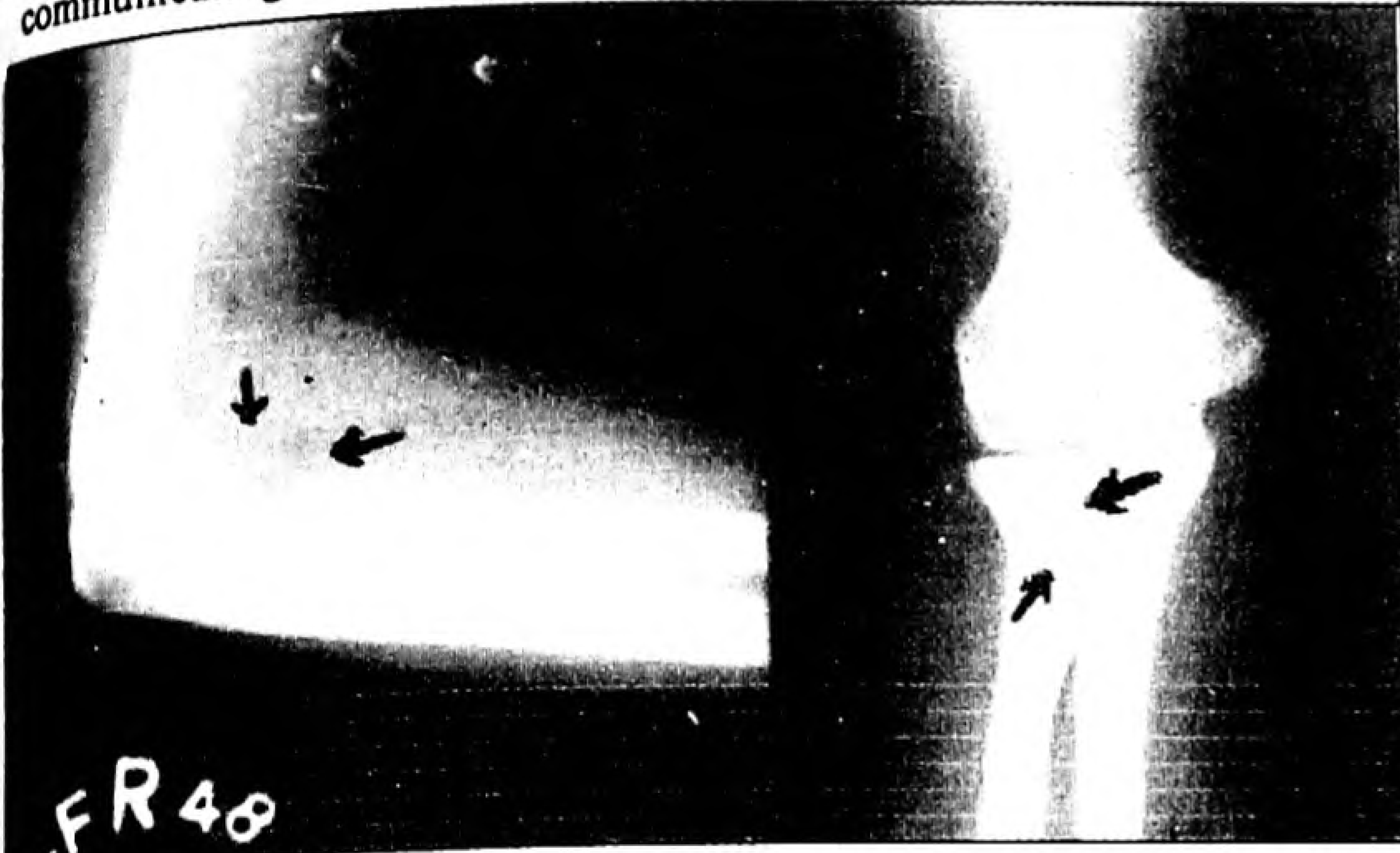

Fig 2: Ganglion of neck of radius communicating with soft tissue ganglion in the cubital fossa

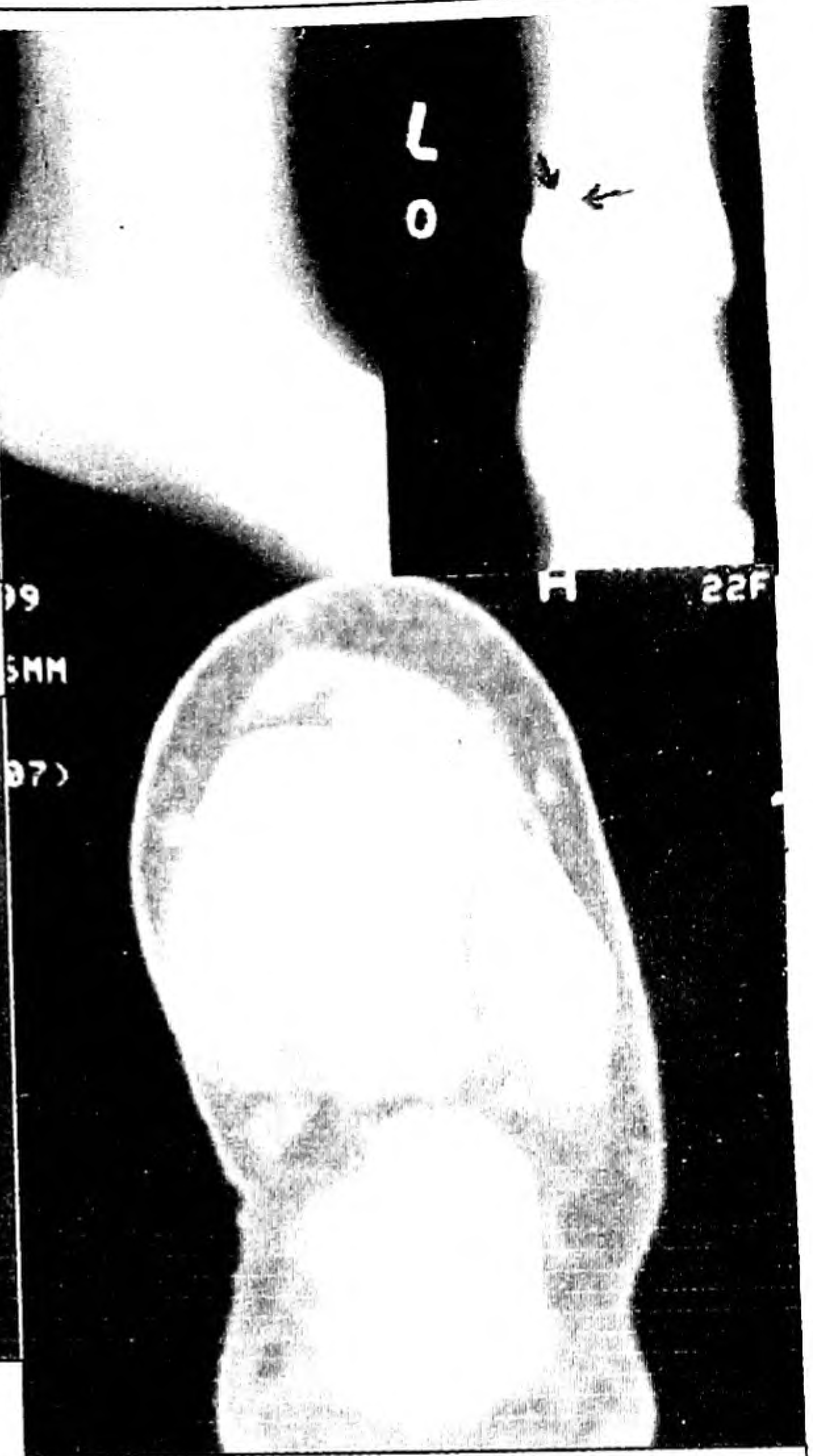

Fig 3: Ganglion in the supramalleolar region of tibia shown on x-ray as well as CT scan.

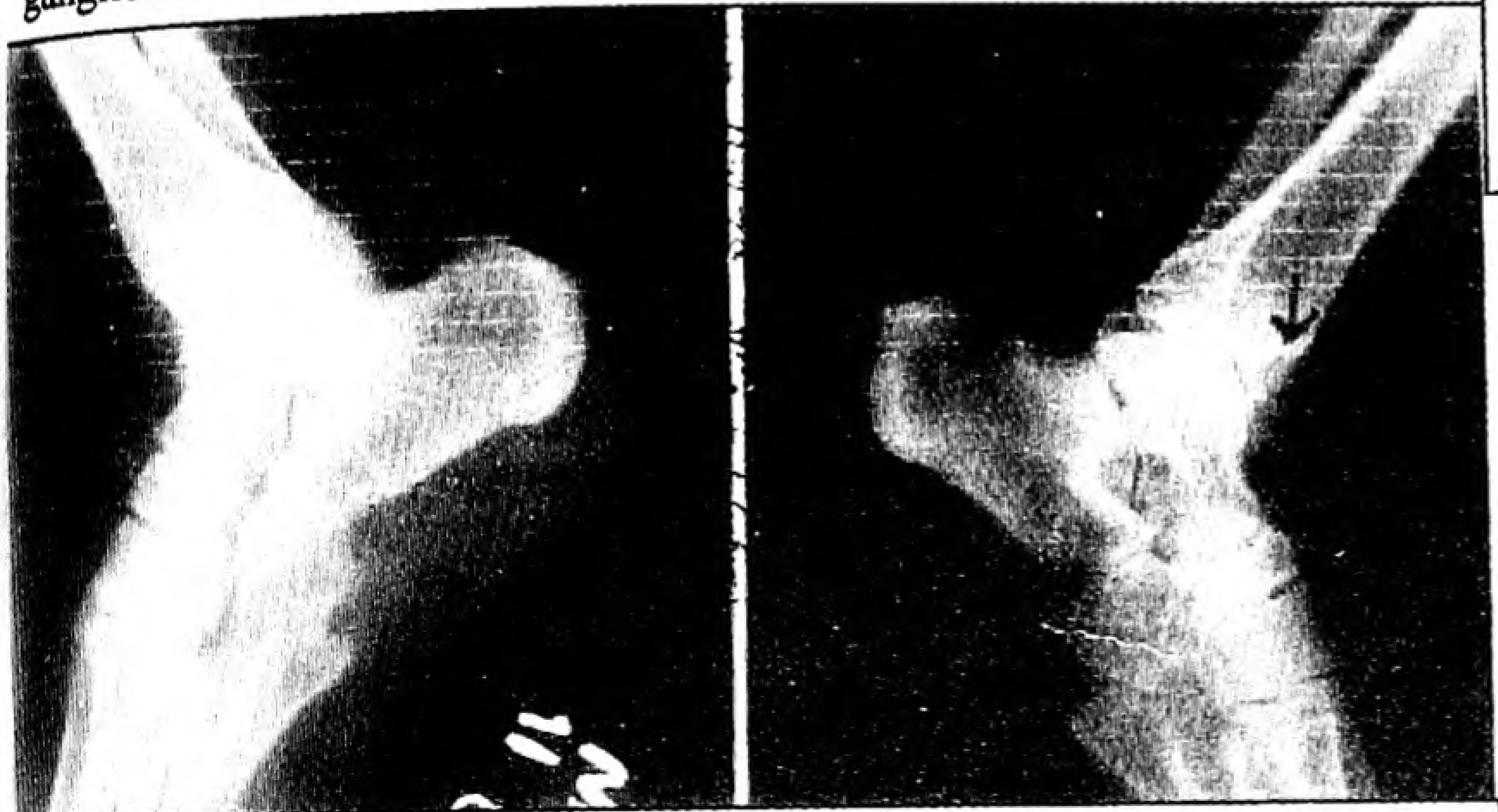

Fig 4: Ganglion of juxtra-articular portion of lower end of tibia anteriorly communicating with soft tissue ganglion along anterior aspect of ankle.

of the ankle showed a subchondral lytic lesion of tibia communicating anteriorly with soft tissue ganglion (Fig 4). Excision of the lesion was done and there was no recurrence of the lesion after 2 years of follow up.

DISCUSSION

Intraosseous ganglia are histologically identical to their soft tissue counterparts. Most commonly they are seen adjacent to the hips, ankles, knee, or wrist and are often mistaken for other neoplasms occurring in the epiphysis[5]. Although soft tissue ganglia are very common, however, intraosseous ganglia are much rarer and typically present with diffuse pain related to an otherwise normal joint in a healthy middle aged patient[6]. On roentgenography, an intraosseous ganglion appears as a well-demarcated circular radiolucent defect surrounded by a band of sclerotic bone. There is no expansion of the overlying cortex or periosteal reaction. Varying degrees of thinning and expansion of the cortex are usually present in giant cell tumours, aneurysmal bone cysts, chondromyxoid fibroma, and fibrous dysplasia. Stippled calcifications may be present in chondroblastoma, chondromyxoid fibroma, and enchondroma, but they have not been described in intraosseous ganglia. The periarticular changes usually seen in Osteoarthritic cysts, synovial cysts, and cysts of rheumatoid arthritis aid in differential diagnosis. Histology confirms the diagnosis and readily differentiates intraosseous ganglia from other lesions having similar x-ray appearance[7] . However, the final diagnosis always remains in the hands of the histopathologist. Although the pathogenesis is unclear, intraosseous ganglia are benign lesions. Simple curettage with or without bone grafting is adequate for persistently symptomatic lesions and recurrence has rarely been reported [8,9] .

Intraosseous ganglion is thought to arise from an intramedullary metaplastic event[9] or is believed to be intraosseous extensions of local soft tissues. Magree (1995) believed intraosseous ganglion of wrists to be more common than previously reported and found bone scanning, conventional radiography, and / or MR imaging to be useful tools in diagnosis of intraosseous ganglion in unexplained wrist pain[10].

There are only limited cases reports of soft tissues ganglia communicating with intraosseous ganglion cyst like that of scaphoid[11], subperiosteal ganglion of the tibia communicating with knee[12], ganglion cysts of the knee with articular communication [13]. All these cases have shown communication, which was demonstrated by urografin injection.

Histologically, the cyst consists of a thin fibrous wall encasing gelatinous material derived from mucoid degeneration. The contents of the cyst are free of necrotic bone and other debris. A synovial lining is not apparent, since neither a distinct layer of epithelial or endothelial cells nor the normal vascularity associated with synovial tissue is present.

The pathogenesis of intraosseous ganglia is obscure. Common soft tissue ganglia are believed to arise from a herniation of the synovium into the fibrous connective tissue surrounding the tendon sheaths and articular capsules. The gross and histological similarity between intraosseous and soft tissue ganglia suggests a common pathogenesis. The evidence of this fact as noted by Crabbe (1966) is the close anatomical proximity of the intraosseous lesions to such ligamentous insertions as the medial collateral ligament of the ankle, cruciate ligament of the knee, and triangular fibrocartilage of the wrist[14].

REFERENCES

1. Kernick CB, Kreipke DL, Davis TE Jr. Intraosseous ganglion of the radial head 11 years after fracture. Orthopaedics 1994 Jan; 17(1): 71-2.
2. Bennett DC, Hauck RM. Intraosseous ganglion of the lunate. Ann Plast Surg 2002 Apr; 48(4): 439- 42.
3. Lorente R, Moreno M, Quiles M. Bilateral intraosseous ganglia of the lunate: a case report. J Hand Surg (Am) 1992 Nov; 17(6): 1084-5.
4. Ferkel RD, Field J, Scherer WP, Berstein ML, Kasimian D. Intraosseous ganglion cysts of the ankle: a report of three cases with long-term follow-up. Foot Ankle Int 1999 Jun; 20(6): 384-8.
5. Pope TL Jr, Fechner RE, Keats TE. Intraosseous ganglion. Report of four cases and review of the literature. Skeletal Radiol 1989; 18(3): 185-7.
6. Gelmann SB, Pacivic B. Multiple intraosseous ganglia in same extremity. NY State J Med 1976; 76: 252-4.
7. Makhija MC, Lopano AJ. Intraosseous ganglion. Bone imaging with Tc^{99m}. Clin Nucl Med 1983; 8: 54-5.
8. Bauer TW, Dorfman HD. Intraosseous ganglion. Am J Surg Pathol 1982; 6: 207-13.
9. Daly PJ, Sim FH, Beabout JW, Unni KK. Intraosseous ganglion cysts. Orthopaedics 1988 Dec; 11(12): 1715-19.
10. Magee TH Rowedder AM, Degnan CG. Intraosseous ganglia of the wrist. Radiology 1995 May; 195(2): 517-20.
11. Kligman M, Roffman M. Urograffin injection demonstrated soft tissue ganglion communication with an intraosseous ganglion cyst. Bull Hosp Jt Dis 1998; 57(2): 96-8.
12. De Maeseneer M, De Boeck H, Shahabpour M, Hoorens A, Oosterlinck D, Van Tiggelen R. Subperiosteal ganglion cyst of the tibia. A communication with the knee demonstrated by delayed arthrography. J Bone Joint Surg Br. 1999 Jul; 81(4): 643-6.
13. Malghem J, Vande berg BC, Lebon C, Lecouvet FE, Maldague BE. Ganglion cysts of the knee: articular communication revealed by delayed radiography and CT after arthrography. AJR Am J Roentgenol 1998 Jun; 170(6): 1579-83.
14. Crabbe, W.A: Intraosseous ganglia of bone. Bri. J. Surg. Jan.1966; 53:15-17.

CHRONIC LOW BACK PAIN - COMPARATIVE ANALYSIS OF TREATMENT RESPONSE TO DRUGS AND DIFFERENT PHYSICAL MODALITIES

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ABSTRACT: On hundred and sixty patients of low back pain of more than three months attending Institute of Medicial Sciences Soura, Srinagar, Kashmir, India were studied. the available data including age, sex, other associated complaints, socioeconomic status, vocational factors and detailed previous and present backache symptoms were analyzed. A thorough physical examination and examination of spine along with relevant investigation were studied. Four groups of forty patients in each group were assigned to four different treatment regimens including moist heat and exercise, combined ILT+MWD and exercise, drugs and exercise, and low intensity SWD (placebo). ILT+MWD and exercises were found superior to other three regimens in the treatment of chronic low back pain.

Key word: *Chronic low back pain, Drugs, Physical Modalities.*

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Introduction

The importance of back pain syndrome is due to their high prevalence in the community and their effect on the individual in terms of pain and disability. Almost 80% of all the people experience back pain to some extent during their active life[1,2] a price paid for uprightposture.

Back pain in most cases is self limiting, with recovery within one month in 70% and within two to three months in 90% while about 4% seem to have a duration exceeding six months[3]. Although the course is usually benign, but the number of severely disabled people is considerable owing to the general occurrence of the ailment, so the problem of chronic low back pain is of great magnitude medically, socially and economically.

The back pain patients, with more than six months incapacity to work have been found comparable in number with those unfit for work due to rheumatoid arthritis and heart ailments[4] with only 50% of patients ultimately return to work[5].

The men and women are equally affected, most commonly between the age of 30 and 55 years[6]. Although the long term disability is much prounced in the older age groups but it is clear that the back pain usually affects men in their most productive life where as women often report low back pain symptoms after the age of 60[7]. The back pain is a clinical manifestation that can be caused by a variety of known diseases and morphological changes. The herniated disc is thought to account for 5% of all the cases[8]. The back pain is also caused by infections, tumours, fractures and osteoporosis, ankylosing spondylitis, rheumatoid arthritis, spinal stenosis and intra-abdominal or intrapelvic diseases. Many of back pain patients do not fall in any of the above mentioned categories and the other associated risk factors including involvement in occupations that require repetitive lifting in forward bend and twisted position, exposure to vibration caused by vehicles or industrial machinary,[10,11] cigarette smoking[12] and obesity should be thought of. Depression, anxiety, hysteria, alcoholism, increased divorce rates and health problems like headache and ulcers are frequently reported in chronic back pain patients[13,14,15].

The low back pain so common a disorder and despite the frequency of this health problem little is known regarding the efficacy of specific treatment regimens. Although the physical modalities are often the treatment of choice, yet very few studies are available regarding the comparative efficacy of different modalties. The study has, analysed chronic low back pain and compared efficacy of different physical modalities in patient outcome.

Material and Methods

This prospective study was carried at Institute of Medical Sciences, Soura, Srinagar, Kashmir, India, 160 patients attending Physical Medicine & Rehabilitation and Internal Medicine departments from January 1989 to December 1991 for low back pain with symptoms for more than three months were studied. In an effort to assemble a group of uncomplicated mechanical back pain, the patients with pregnancy, back surgery, spondylolisthesis, tumours, fractures, infections, senile osteoporosis and structural

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scoliosis were excluded.

All the available data including age, sex, other associated complaints, socioeconomic and vocational factors with complete questionnaire about previous and current back symptoms were made. All the patients taken in the study were subjected to a thorough physical examination with special stress on the examination of spine. the patients were subjected to routine investigations including hematological and biochemical tests with radiography of chest, lumbosacral spine and other investigations like Reheumatoid factor, ANA, HLA-B27, bone san, myelography and CT Scan wherever needed. On initial assessment the history of back pain was summarized and the duration of the current episode was recorded. The intensity and quality of pain and degree of functional limitation were analyzed in the following way. The patients were asked to choose one of the following descriptions of their pain.

1. Terrible pain.
2. Intense pain
3. Sharp stabbing pain
4. Spasmodic pain
5. Intense dull ache
6. Slight stabbing pain
7. Slight dull ache
8. Feeling of discomfort
9. Feeling of stiffness
10. Feeling of exhaustion.

The numbers 1 to 5 were pooled into one group under the designation "intense pain" and remaining as "slight pain".

To assess the functional limitations, patients were asked about 10 different daily activities like walking, walking in stairs, running, stooping over a wash basin, carrying a bag, making a bed, riding in a car, going to the market, participating in sports, sitting for a longer period of time and are asked whether they could perform it and if so whether with or without pain aggravation. The points to three possible answers (No difficuluties, painful but possible, not possible owing to pain) were recorded. From the above observations the "pain index" was formulated to ascertain the intensity of pain. The range of the "pain index" varies from 0-70 points (0-No pain and 70 - maximum pain).

Question regarding the "pain index"

Symptoms	No	Yes
Intensive pain	0	15
Sleep disturbing pain	0	5
Continuous pain	0	5
Increased pain when coughing and sneezing	0	5
Increased pain when flexing back	0	10
Increased pain when extending back	0	10
Functional limitation (10 questions each ranging from 0-3 points)	0	30

The localization of the pain was plotted on a graph. The presence of postural scoliosis, muscle spasm and pain on palpation and percussion were noted. The range of motion and aggravation of pain in different planes of motion were registered. Flexion, lateral flexion and extension were measured by Schober's modified method as modified by Moll and Wright 1971[16].

Flexion was measured by plotting marks on the lumbosacral junction and ten centimeters above and five centimeters below the lumbosacral junction. The distance between the upper and lower marks were measured in standing erect and then in maximal flexion. The difference between the distances represents the ability to flex the spine.

Lateral flexion was measured by plotting one mark on the iliac crest and other mark on a horizontal line through the xiphoid process in the mid axillary line of the trunk. The distance between the two marks were measured in standing erect and in maximal lateral flexion. The diffference between the distances represented the lateral flexion of the spine. Extension was measured by using the same marks as in lateral flexion. A thread with a pointed weight was held so that the point of the weight coincided with the lower mark. The patient was then asked to bend backwards as much as possible. The pointed weight traversed the flank. The distance measured represented spinal extension; rotation was measured with a gomometer with the patient sitting.

The patients were randomly assigned to one of the four treatment regimens.

Group A- (40 Patients) Moist heat and exercises

Group B-(40 Patients) Combined therapy of intermittent lumber traction with MWD and exercises.

Group C-(40 Patients) Drugs and exercises

Group D-(40 Patients) Placebo (Low intensity SWD)

The moist heat was given daily for 20minutes with hydro collator packs preheated to a temperature of 150 degree Fahrenheit. These hydro collator packs are enclosed packs of silica gel, which are heated in a stainless steel cabinet having a thermostat up to 160 degree Fahrenheit. These packs maintain heat for longer period and convienently used for 20-30 minutes treatment periods without reheating. The packs are wrapped in several layers of towel to provide uniform distribution of heat and prevent skin burns.

The combination therapy of intermittent lumber traction (ILT) with Micro wave diathermy (MWD) was given with traction intermission programme between 5 & 20 seconds and on a average 12 to 18 kgs of traction weight was applied except for obese and overweight patients, where the maximum traction weight applied was up to 1/3rd of total body weight. The MWD given has penetration intensity beyond sub dermis up to the bone depending upon the applied intensity. The total therapy time was 20 minutes.

In Group C drugs used were mostly NSAID's of which Ibuprofen, Diclofenac sodium, Salicylates, Indomethacin, and Acetaminophen were prescribed with Diazepam whenever needed. The drugs were given by mouth in recommended therapeutic doses. The selection of the drugs was done on the patient'scompliance.

To the Group D patients short wave of lowest intensity was given. The heat produced at this intensity may at the most increase local blood flow of the subcutaneous tissue,do the therapy was regarded as a placebo. Exercises in all the three treatment regimens were same whcih included crook lying, pelvic tucking, and restricted quadriceps and hamstring stretch exercises. The exercises were started gradually untill all the exercises were performed by the patients.

The treatment protocols were prescribed for 10 days (5 days per week) per month for a period of 3 months and during the intervening period and after the completion of intervention therapy the patients were encouraged to continue the exercises at home usually different technique but on same basic principle to sustain the beneficial effects of intervention therapy.

Observation and Discussion

The study patients consisted of 91 males (Age range 25-61, median 40.89 years) and 69 females (Age range 16-64 years median 38.58 years). 92(57.5%) patients were from lower middle class and 41(25.6%) were from upper middle class with 19(11.8%) from lower class and only 8(5%) patients were from upper socioeconomic group.

There were other associated complaints like chronic duodenal ulcer in 6 patients. Two patients were suffering from irritable bowel syndrome where as two patients each had cervical spondylitis, adhesive capsulitis, osteoarthritis knee and fibroparenchymal chest lesions. The evaluation of risk factors revealed that 69(43.1%) subjects had psychological symptoms in the form of anxiety or depression. The time between onset of back pain and the initial assessment of patients ranged from 3 months to 8 years (median 13 months), straight leg raising test was positive in 52% of patients while only 14% had positive below 60 degree. Almost 90% of the patients had pain on lumber flexion. The mean values of extension and lateral flexion were within normal limits and a minority of the patients experienced pain during these movements. Pain on percussion was noted in 38% and tenderness on palpation in 26% of the patients. Less than 20% of the patients had a postural scoliosis where as about 28% patients had prounced muscle spasm. In this study an attempt was made to acheive homogenecity of the diagnosis in the studied group by only including patients with.

1. Pain in lumber region with or without radiation to the thigh.
2. Duration of symptoms more than three months.
3. Exclusion of patients with definite disease entities.

However nothing can be said with certainty about the homogenecity of underlying causes of the pain in the studied group because of knowledge about the etiology of back pain also makes it difficult to find objective methods of assessing clinical manifestations. Thus the clincial methods available for measuring the mobility of the spine or the strength of certain muscles lack normal values. Further more when pain is present the difficulties in obtaining objective values increase, since the investigator obtains the patient's own assessment of the pain intensity and functional limitations rather than measurement of mobility and strength.

In this study, our results fail to demonstrate significant differences between the different treatment approaches on all outcome parameters. There is a marginal trend indicating that the longer the time elapsing between the onset of symptoms and start of therapy, longer is the time taken for recovery. A pronounced difference between Group D (Placebo) and other three groups were demonstrated. The patients of Group B (combined ILT+MWD & Exercise) especially with positive SLR test and radicular pain showed a faster recovery with increase in range of movements of spine especially forward flexion, the patients with marked postural scoliosis showed a rapid initial improvement with moist heat and exercises, however the results did not reveal any marked differences between Group C (Drugs and exercises) and placebo group. Hurwitz et al[17] also stated that the physical modalities used by chiropractors in the managed care organization did not appear effective in the treatment of patients with low back pain, although a small short term benefit for some patients could not be ruled out.

Linda and Bombarier[18] acknowledged the trend of using electrotherapeutic and thermal modalities in management of chronic low back pain with uncertain effectiveness. The poor evidence of efficacy existing for the use of different physical modalities in treatment of patients of chronic non specific low back pain has also been stated by Nordin and Compello[19], Ernst and Fialka[20] also emphasized the definitive assessment of present employed physical treatment modalities as enough controlled trails have not been carried out.

The study concluded that combined therapy of intermittent lumber traction with Microwave diathermy and exercises for the treatment of chronic low back pain is superior to other three regimes and none the other three therapeutic regimes are symptomatically superior to one another.

References:

1. Hult L. cervical, dorsal and lumber spine syndromes. Acta orthop. Scand 1954 Suppl 7.
2. Nachemson A.I. The lumber spine, an orthopaedic challenge.

Spine 1976;1:1,59-71.

3. Horal J. The clincial appearance of low back pain disorders in the city of Gothenburg, Sweden. Comparison of incapacitated probands with matched controls. Acta orthop. Scand 1969;Suppl. 18.
4. Heber L.D. Disablign effects of chronic diseases and impairment. J. Chronic Dis 1971;24:469-487.
5. McGill C.M. Industrial back problems - a control programme. J. Occup. Med. 1968;10(4):174-178.
6. Kelsey J.L. An epidemiological study of acute herniated lumber intervertebral discs. Rheumator rehabil 1975;14-159.
7. Biering Sorensen F. Physical measurements as risk indicators of low back trouble over a one year period. Spine 1984;9:106-119.
8. Hirsh C. Etiology and pathogenesis of low back pain. Israel J. Med. Sci 1966;2(3):362-370.
9. Damkot D.K. et al. The relationship between work history, work environment and low back pain in men. Spine 1984;9(4):395-399.
10. Frymoyer J.W. et al. Risk factors in low back pain. J. Bone Joint Surg. 1983;65-A(2):213-218.
11. Kelsey J.L. and Hardy R.J. Driving of motor vehicles as risk factor for acute herniated lumber intervertebral discs. American J. Epidemo 1975;102(1):63-73.
12. Kelsey J.L. et al. Acute prolapsed lumber intervertebral disc - an epidemiological study with special reference to driving automobiles and cigarette smoking. Spine 1984;9(6):608-613.
13. Anderson J.B.J. et al. The intensity of work recovery in low back pain. Spine 1983;880-884.
14. Frymoyer J.W. et al. Psychological factors in low back pain disability. Clin. Orthop. 1985;195:178-184.
15. Vallfors E. Acute, sub acute and chronic low back pain - Clinical Symptoms, absenteeism and working environment. Sacand. J. Rehabil Med (Suppl) 1985;11:1-98.
16. Moll J.M.H., Wright V. Normal range of spinal mobility. Ann. Rheum. Dis. 1971;30:381-386.
17. Hurwitz E.L. The effectiveness of physical modalities among patients with low back pain randomized to chiropractic care: findings from the UCLA low back pain study. J Manipulative Physiol Ther 2002;25(1):10-20.
18. Li LC, bombardier C. Physical therapy management of low back pain: an exploratory survey of therapist approaches. Physical. Ther 2001;81(4):1018-28.
19. Nordin M, Campello M. Physical therapy: exercises and the modlities: when, what, and Why? Neurol Clin 1999;17(1):75-89.
20. Ernst E, Fialka V. Conservative therapy of backache. Part 5: TENS, acupuncture, biofeedback, traction, cryotherapy, massage and ultrasound. Fortschr Med 1993;30;111(27):420-2.

SURVEILLANCE OF VACCINE PREVENTABLE DISEASES IN THREE BLOCKS OF GOVT MEDICAL COLLEGE SRINAGAR

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Abstract: *We could identify only three cases of tuberculosis accounting for 0.12% of total children, 194 cases of pertusis accounting for 7.76% of total children. Of the total 28 measles cases the enquiry revealed that only 7 cases were immunized and the remaining 21 cases unimmunized. Of the total children surveyed, 99.66% had received measles vaccine. Among partially immunized children, 69.19% were not affected at all. It was noted that only 3 suspected cases of acute flaccid paralysis were found from the three blocks under study which were latter classified as non-polio.*

Key words: *vaccine preventable diseases, measles, pertusis, immunization coverage*

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INTRODUCTION;

India has large number of children, with 72 babies dying out of every 1000 babies born in a year.[1] Diseases of early childhood, preventable by vaccination remains a serious problem in developing countries despite the continuing increase in proportion of children receiving vaccines[2]. Vaccine preventable disease are decreasing with NNT Elimination (Neonatal tetanus) in near future. Measles incidence decreased since 1992 and polio is expected to be eradicated by 2005 AD with the help of pulse polio immunization. In 1993, Global Programs on vaccine (GPV) was launched to sustain high levels of immunization coverage, develop good surveillance network for VPD (Vaccine Preventable Diseases) and evolve eradication strategy. Success in UIP (Universal Immunization Programme) Led to launch of CSSM (Child Survival and Safe Motherhood) Program in 1992 and RCH (Reproductive Child Health) program in 1997 incorporating immunization as one of the major components[3].

Each year around 12 million children aged <5 years die in resource poor countries and 70% of thee deaths are due to communicable diseases and or due to malnutrition. Since mid-1900 the WHO has been leading the development of an integrated approach to care for ill children at the primary care level, a programme known as integrated management of child hood illness. This approach combines improved management of childhood illnesses (IMCI) with aspects of nutrition, immunization, and maternal health. This programme replaces or complements a number of vertical programme that were aimed at specific groups of conditions including Control of Diarrhoeal Diseases (CDD), acute respiratory infections (ARI) and expand the immunization programme (EPI). This approach has many advantages because it is well accepted by pediatricians in tropical countries and also because it conforms to secondary care practice.[4]

Methodology

While assessing the implementation programme of immunization being in force at state and national level, an attempt has been made to review the success in relation to its acceptability, concept, and incidence of its related diseases. With this in mind, immunization assessment has been carried out in the field practice areas of govt. medical college Srinagar. It is a purely a sample based study and the enquiry has been restricted to children under five years of age. For this purpose, a stratified random sample of 12 villages has been drawn from all the villages falling within the jurisdiction of three medical blocks namely Hazratbal, Ganderbal, and Lar. The frame constituted two lists of villages in hilly and plain areas separately. With this stratification, four villages have been selected at random from each block, using two and three digit tables, two villages from hilly, and two from plain areas separately. Four proformas were initially devised to undertake the study, before being introduced in the field they were pretested, and necessary modification made wherever need arose. The reference period of the study being from 1st April 2000 to March 2001. The magnitude of VPD (vaccine preventable disease) was also recorded. A case definition for vaccine preventable diseases was used.[5]

CLINICAL DIAGNOSTIC CRITERIA:

DISEASE	CLINICAL CASE DEFINITION
Diphtheria:	Illness characterized by laryngitis or

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Table 1: Immunization status in surveyed areas.

Age in years	Children immunized											
	PARTIALLY						FULLY					
	Males		Females		Total		Males		Females		Total	
	No.	%	No.	%	No.	%	No	%	No.	%	No.	%
0-1.5	68	18.3	120	35.1	188	26.4.	303	81.7	222	64.9	525	73.6
1.5-2.5	85	27.6	87	30.5	172	29.0	223	72.4	198	69.5	421	71.0
2.5-3.5	71	27.6	98	43.4	169	35.0	186	72.4	128	56.6	314	65.0
3.5-4.5	63	29.4	106	59.6	169	43.1	151	70.6	72	40.4	223	56.9
4.5-5	38	21.1	17	12.3	55	17.3	142	78.9	121	87.7	263	82.7
Total	**325**	**24.4**	**428**	**36.6**	**753**	**30.1**	**1005**	**75.6**	**741**	**63.4**	**1746**	**69.9**

Note: Fully children stands age appropriateness of children covered under OPV, DPT, BCG, and measles.

pharyngitis or tonsillitis and adherent membrane of tonsil, pharynx, or nose

Measles: Any person with fever and maculopaular rash and cough, coryza and conjunctivitis.

Pertusis: Any person with coughs lasting at least for two weeks with one of the following:

- Paroxysms of cough
- Inspiratory whoop.
- Vomiting immediately after coughing without other apparent cause

Poliomyelitis: Any child less than 15 years of age with AFP (acute flaccid paralysis) or any person with paralytic illness when polio is suspected other causes being excluded like Trauma, Guillein Barre Syndrome etc.

Neonatal Tetanus: *Suspect:* - any neonatal death between 3 and 28 days of age in which cause of death is unknown or any neonate reported as having neonatal Tetanus but not investigated.

Confirmed: Any neonate with a normal ability to suck and cry during the first two days of life, who between 3 and 28 days of life cannot suck normally

Since clinical case definition of tuberculosis is difficult because there can be silent infection as we as varied clinical picture so, tuberculosis children whenever suspected were got confirmed and investigated at Govt. Children Hospital by a pediatrician. As for as poliomyelitis is concerned, for confirmation approximately 8-10 gms of two stool samples were collected 24-48 hours apart, within 14 days of onset of paralysis and were transported to the regional testing laboratory i.e. Central Research Institute Kasuli Himachal Pardesh. The help of WHO surveillance officer was taken in this regard. A 60-day follow up was made to see for residual paralysis. Surveillance units were set up at block headquarters with doctor available. Since most of the centers remain closed after 4 pm , hence a list of practicing doctors in the study area was made. They were requested to report by telephonic measures or through personal contact the patients detected as being VPD if they fall from the study area. The data was supplemented by surveillance from the referral hospital namely Govt. pediatric hospital. The resident doctors were requested to intimate any VPD from the study area.

Observations:

The population of the 12 villages sampled totaled to 17,063 comprising of 1772 house holds which gives an average of 7.6 members per house hold Number of children less than 5 years old per family averaged 1.4. This needs to be mentioned here that all the selected villages are predominantly rural but appears to be well equipped in relation to the available facilities. All the selected villages have approach to the health unit and AWC are functioning in all these areas. In all 2499 children, less than 5 years of age have been surveyed during the reference period of which 53% were males and 47% females. The immunization status is given in **table 1**.

As far as vaccine preventable diseases are concerned, we could identify only three cases of tuberculosis accounting for 0.12% of total children, 194 cases of pertusis accounting for 7.76% of total children. As regards measles only 28 cases could be identified which accounted for only 1.12 % the specific details in relation to age and sex is shown in **table 2.**

The situation necessitated to asses the significance of these vaccines in relation to the affected cases. This has been shown in **table 3,** which shows efficacy of measles vaccine in children < 5 years age. Of the total 28 measles cases the enquiry revealed that only 7 cases were immunized and the remaining 21 cases unimmunized. Of the total children surveyed, 99.66% had received measles vaccine.. Among unimmunized children, 69.19% were not affected at all. As such chi-square test has been applied with the view to assess the efficacy of vaccinations which depicted highly significant results at $p<0.001$. As such, it was inferred

Table 2. Incidence of vaccine preventable disease in children bellows 5 years.

Vaccine preventable disease	Sex		Age in years 0-1.5	1.5-2.5	2.5-3.5	3.5-4.5	4.5-5	Total
Tuberculosis	Male	No.	-	-	—	2	-	2
		%	-	-	-	0.93	-	0.15
	Females	No.	—	-	-	1	1	
		%	-	-	-	-	0.72	0.09
	Total	No.	-	-	-	2	1	3
		%	-	-	-	0.51	0.13	0.12
Pertusis	*Males*	*No.*	*21*	*29*	*17*	*39*	*31*	*137*
		%	5.66	9.42	6.61	18.22	17.22	10.30
	Females	No.	9	17	14	11	6	57
		%	2.63	5.96	6.19	6.18	4.35	4.88
	Total	No.	30	46	31	50	37	194
		%	4.21	7.76	6.42	12.76	11.64	7.76
Measles	Males	No.	4	3	4	-	2	13
		%	1.08	0.97	1.56	-	1.11	0.98
	Females	No.	6	5	2	1	1	15
		%	1.75	1.75	0.88	0.56	0.72	1.28
	Total	No.	10	8	6	1	3	28
		%	1.40	1.35	1.24	0.26	0.94	1.12

Note: *(-) dash stands far nil.*

that vaccine is very much effective against the disease.

TABLE 3. Significance of measles vaccine in children bellow 5 years

Immunization status	Children below 5 years Affected No.	%	Not Affected No.	%	Total
Immunized	7	0.34	2069	99.66	2076
Unimmunized	21	32.81	43	67.19	64
Total	28	1.31	2112	98.69	2140

Note: *of the total children 359 children were not eligible for immunization being bellow 9 months as such these have not been included and no one suffered from measles Chi-square (x^2): 495.43*

P value: <0.001 highly significant

Results: *The chi-square test applied for assessing the relation between the attributes depicts highly significant results at $p < 0.001$ and as such it is concluded that the vaccine is very much effective against the disease.*

Like wise significance of the DPT, vaccination in children suffering from pertusis has also been evaluated and placed in **table 4**. Of the total immunized 96.99% of the cases were not affected at all and in 19.89% of unimmunized children disuses was seen. Similar treatment of chi-square test ahs been applied with the view to assess the efficacy of vaccinations which depicted highly significant results. As such, it was inferred that vaccine is very much effective in preventing and controlling pertusis.

TABLE 4. Significance of DPT vaccine on pertusis in children < 5 years of age.

Immunization status	Children below 5 years Affected No.	%	Not Affected No.	%	Total
Immunized	54	3.01	1741	96.99	1795
Unimmunized	140	19.89	564	80.11	704
Total	194	7.76	2305	92.24	2499

Chi-square (x^2): 201.20

P value: <0.001 highly significant

Results: *The chi-square test applied for assessing the relation between the attributes depicts highly significant results at $p < 0.001$ and as such it is concluded that the vaccine is very much effective against the disease.*

As for as acute flaccid paralysis (AFP) cases is concerned, it was noted that only 3 suspected cases from the three blocks under study have been reported irrespective of the present study. The stool samples of these cases were collected and sent for wild virus isolation bur the results were contradictory. A 60 day follow up of these cases was made and no residual paralysis cold be ascertained.

Discussion:

Kashmir is situated entirely in the Himalayan region and is northern most state of the country. It is a cold region with the seasonal temperatures varying from -10°C during winter and 35°C in summers. The present study was undertaken in all the three blocks representing the field practice areas of the Govt. Medical College Srinagar namely block Hazratbal, Ganderbal and Lar. These blocks have village strength of 128, 96, and 55 with the population of 98108; 91503 and 49993 respectively. Out of which 12 villages have been randomly selected with a population of 17063 that makes about 7.11% of total study population. About 69.9% of the children in the experimental area were fully immunized, 30.1 % of children in the age group of 0-5 years were found to be partially immunized. No unimmunized child was noticed in the experimental area. As per NFHS-2, (6) survey the figures for J&K for fully immunized children stands at about 60-79%. The absence of non- immunized children in the study area speaks about the health awareness of people boosted by the NID's campaign. This campaign has also given our health workers an opportunity to advocate routine immunization.

As for as vaccine preventable diseases are concerned, the present study found only three cases of tuberculosis in the age group selected which accounts for about 0.12%. Rao and Bharmbe MS (7) detected about 20 such cases in infancy and about 266 cases in the age group of 1-14 years in a hospital based study carried out from year 1982-86. The low percentage of tuberculosis patients in the present study population can be explained because of difficulty in diagnosing the disease especially in initial stages or because of varied clinical presentation of tuberculosis in children.

About 196 cases of pertusis were detected in the experimental area that accounted for about 7.76%. Rao and Bharmbe MS (7) detected about 11 such cases in a hospital-based study. As per WHO report on vaccine preventable diseases (8), about 11,264 cases of pertusis were reported from India for the year 1999. More cases in the study area proportionate to the national figures can be explained because of high dropout rate of DPT booster.

Twenty-eight cases of measles were reported from the study area that accounted for about 1.12% of the study population. Rao and Bharmbe MS (1991) detected about 111 such cases in a hospital-based study. As per WHO report on vaccine preventable diseases (2000), about 21,013 cases of measles were reported from India for the year 1999.

As for as acute flaccid paralysis is concerned (AFP) only three such cases were detected in the study blocks irrespective of the study villages. Confirmation of these showed them to be non-polio in origin. The 60-day follow up of these cases also proved them to be non-polio. For the year 1999, 1126 cases of wild poliovirus were detected from India mostly from UP and Bihar. The number decreased to about 263 wild poliovirus cases for the year 2000. 80 cases of AFP were detected from J&K in the year 2000, out of which 79 were non-polio and only one case was pending for confirmation (9).

The figures with measles and pertusis called for checking the efficacy of vaccines DPT and Measles in controlling these diseases. Both of these were found to be highly effective in preventing the diseases. Kandpal SD (10) found that 96.50% of cases that were immunized with measles were Seropositive for the antibody titer, while as only 3.5% were Seronegative. This shows the efficacy of the vaccine in preventing the disease. Bhandari R et al (11) reported that three doses of DPT produced a high seroconversion rate of antibody titer against diphtheria, pertusis, and tetanus.

No case of diphtheria and neonatal tetanus was detected from the study area. As per WHO report on vaccine preventable diseases (2000), about 1786 of diphtheria and 610 cases of neonatal tetanus were reported from India for the year 1999. Nil case in our study speak about the efficacy of DPT in preventing diphtheria. This could be also due to high herd immunity. The high percentage of TT2/booster coverage for pregnant woman as well as safe delivery practices could be the reason for nil case of neonatal tetanus.

CONCLUSION

Vaccine preventable diseases although decreasing yet in certain areas they are still a public health problem. What is needed is an effective surveillance for the same as well as improving the immunization coverage so that effective measures are adopted.

Bibliography

1. Anonymous " Annual report 1999-2000, Ministry of Health and Family welfare Govt Of India New Delhi
2. Sabin A. " Strategy for rapid elimination and continuing control of poliomyelitis and other vaccine preventable diseases in children in developing countries" Brit Med J 1986,292:531-533
3. Parthsathy A. U.I.P. IAP Textbook of Paed.: 1999 Ed. (167-169)
4. Anonymous. "Immunization in childhood" 'Annales Nestle': 2000; 58: 106
5. Vaccine By Plot kin 1999 Edition ,1058, W B Saunders
6. NFHS-2 (1998-99) survey Health monitor 1999; foundations of research in Health system, Azad society Ahamdabad
7. Rao SP and Bharmbe MS "Vaccine preventable diseases in eastern Mahrashtra- A hospital based Analysis Indian Pediatrics; "1991;28:629-633
8. WHO Vaccine Preventable Diseases Monitoring System; 2000 Global Summary WHO-2000
9. AFP Alert NPSP-GOI ;2001; Jan-Feb; vol 5
10. Kandpal SD, Malik A and Khan Z "Measles antibody titer in 0-5 years children at Aligarh Indian J. of Comm. Medicine 1999; 24:115-119.
11. Bhandhari B; Nagori G; Mandowara SL. "Immunization Status in ICDS blocks Ghari." Ind. Paed. 1981; 18: 187-192.

NEPHRONOPHTHIS AND NEPHROLITHIASIS - AN UNCOMMOON ASSOCIAITON

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Abstract: Nephronophthisis and medullary cystic disease constitute 2-10% f the hereditary renal diseases. On genetic basis Nephronophthisis can be infantile, juveile or adolescent. Only familial juvenile nephronoophthisis is associated with an extra renal involvement. However, no association with nephrolithiasis has been mentioned in English literature. Here we present a case of juvenile nephronophthisis with extra renal involvement an nephrolithiasis.

Key words: Medullary Cystic disease, nephronophthisis, tapeto retinal degeneration, cerebellar degeneration, cone shaped epiphysis, nephrolithiasis.

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Introduction

Nephronophthisis (NPH) and medullary cystic disease (MCD) represent a disease complex with genetic heterogeeity, but have also been discussed as separate entities. This complex was Ist described by Fanconi in 1951 and fewer than 300 cases have been reported by 1980. NPH is responsible for at least 10% of renal failure in children and NPH and MCD combined together account for 4% of all hereditary renal disorders.[1] NPH is an autosomal recessive disorder and can be an infantile, juvenile or adolescent form while MCD is an autosomal dominant disorder and ooccurs in adulthood. Only juvenile form i.e. familial juvenile nephronophthisis is associated with an extra renal involvement i.e. retinal defects, skeletal abnormalities, aminoacidurias, hepatic firbrosis and lesions of central nervous system.

Case Report

RK, a 14 years old female, non-diabetic, non hypertensive was admitted with the complaints of polyuria and polydipsia; diminution of vision; anaemia and Weakness; difficulty in walking; passage of crystal like material in urine and sudden onset of acute breathlessness. On eliciting the history from parents, she was a daughter of a consanguinous marriage with delayed milestones and started with the symptoms of polyuria and polydipsia at an age of 2 years. It was associated with an increase in craving for salts and salty foods. She used to take an excess amount of salt in food than any other family member. Again at the age of 2-3 years, she had difficulty in vision which was progressive in nature and vision got deteriorated to the extent of finger counting in right eye and was almost blind by left eye at the time of admission. At the age of 8 years she used to have generalized body aches and pain in bones & joints. This was followed by difficulty in walking and a sense of imbalance and falling towards the left side more as compared to the right side whenever she used to close her eyes. She used to walk with wide based gait and was not able to run.. For the last 2 years she gave history of passing stones in the urine about four such episodes, which were crystal like white in colour and not associated with hematuria or pain. On examination, weight and height were below 3rd percentile and BMI was 13 kg/m2. There was marked degree of pallor annd horizontal nystagmus with half and half nails signifying chronic renal disease. Pulse was 84/mt. regular and good volume and BP in right arm was 120/84mmhg. There was no abnormality in chest, CVS and abdomen. Apart from generalized wasting, gait and coordination abnormality, central nervous system examination did not reveal anything. In coordination abnormality, there was dysdiadokonesi and impairment of finger nose test heel shin test on left side more as compared to right side. She used to walk with a wide based gait with swaying towards either side more towards the left.

On inestigations Hb-8gm%: TLC=12,000/ cmm: P70L26E2M2: Urea - 289gm/dl: creatinine - 7.6mg/dl: sr. uricacid=6.9mg/dl: B. sugar-110mg/dl: ABG showed metabolic acidosis. Serum electrolytes are shown in Table (1). Urine showed albumin (+): sugar - nil: puscells - 2-3/ HPF:RBC-0-1/HPF: Urine PH-6.0 and specific gravity - 1.005. Urinary electrolyte are showen Table (2).

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Table-1:

Electrolyte	Serum	Normal
Sodium	123meq/lt.	135-150 meq/lt.
Potassium	3.0 meq/lt.	3.5-5.5 meq/lt.
Calcium	7.2 mq/dl.	8-10 mq/dl.
Magnesium	1.4 mg/dl.	1.7-2.6 mg/dl.
Phosphorous	6.2 mg/dl.	3.5 mg/dl.

Table -2:

Electrolyte	Urine	Normal
Sodium	630 mmol/day	100-260 mmol/day
Potassiu	75 mmol/day	25-100 mmol//day
Calcium	1350mg/day	<300 mg/day
Magnesium	1.8mg/dl.	1-13 mg/day
Phosphorous	6.5 mg/dl.	20-60 mg/dl.

Stone Analysis revealed noo uric acid but crystals of calcium, citrate and few cystine crystals. X-ray of the hands showed cone shaped epiphysis in phalanges[1] and plain x-ray abdomen showed bilateral calculi in kidneys..[2] Usg Abdomen showed bilateral small kidneys (Rt 5.7 x 2.4 & Lt. 5.1 x 2.3 cm) with multiple renal stones and poor definition of corticomedullary - junction.[3,4] MRI of the brain showed early features of cerebellar degeneration[5,6,7,8,9] and Retinal examination revealed bilateral retinal degeeration.[11,12,13,14]

Discussion

Nephronophthisis is characterized by a chronic diffuse interstitial nephritis with few or no inflammatory cells and the presence of cysts in the medulla. The presence of these cysts has given rise to the term medullary cystic disease in American literature[2] whereas Fanconi et al (1951)[3] emphasized the atrophy of renal tubules and parenchyma and proposed the term 'Nephronophthisis: However, some authors have proposed the term 'medullary cystic disease' for the disease occurring in adults with a dominant inheritance and the term 'nephronophthisis' for the recessive forms, which seem by far the most common[4].

Delayed onset of polyuria after birth excludes the diagnosis of nephrogenic diabetes insipidus, but polyuria after birth excludes the diagnosis of nephrogenic of normal renal function. Most children have a decreased glomerular filtration rate at presentation and the associated symptom (hyposthenuria and normal urinalysis) tend to suggest disorders such as renal hypoplasia and urinary tract malformations which are the frequent causes of renal insufficiency in children. These diseases have very similar symptoms but more often produce signs of abnormal development during first year of life. Radiological investigation excludes these renal disorders. Practically the presence of renal insufficiency with marked polyuria in absence of haematuria or proteinuria, with a normal urinary tract and kidneys of normal or reduced size occurring in patients aged from 3 to 15 or 18 years is highly suggestive of nephronophthisis..[5]

The desrfiption of nephronophthisis in the literature is confusing since they include real diseases with various clinical features: different modes of inheritance and associated with various disorders. Eye association with nephronophthisis has been estimated as 40 percent of published cases. Early blindness due to cloboma, cataract, retinitis pigmentosa of various origins or even simple anomalies without extinction of the electroretinographic tracings have been reported as 'Seniors Syndrome' or Senior Loken Syndrome.[6]

Similarly, associaton of nephronophthisis with retinal degeneration resembling lebers' Amaurosis, cone shaped epiphysis and cerebellar ataxia have been tered as 'Mainzer - Saldino Syndrome[7,8] while the association of Nephronophthisis with liver fibrosis is called 'Boichis Syndrome[9] and may occur in combination with deafness, cereballar ataxia and tapeto retinal degeneration[10]. Association of nephronophthisis and retinal degeneration in association with asphyxiating thoracic dystrophy have been termed as 'Jeunes Syndrome'. The association of nephrolithiasis with juvenile nephronophthisis has not been mentioned in English literature. However, in addition to nephronophthisis, our patient had retinal degeneration, cerebellar ataxia, cone shaped epiphysis and nephrolithiasis. (The unusual presence of renal stoes in this disorder along with other extra renal organ involvement should make one to rename it as a separate syndrome).

References

1. Klernknecht C: - Inheritance of Nephronophthisis. In topics in Renal medicine: Inheritance of kidney and urinary tract diseases, edited by Spitzer A, Avner ED, Boston, Kluwer Academic Pulbishers PP, 1989;277-294.
2. Smith C.H and Graham, J.B. Congenital Medullary cysts of the kidneys with severe refractory anemia. Am J of disease of children, 1945;69:370-8.
3. Fanconi, G., et al. Die familiare juvenile Nephronophthisis. Helvetica Paediatrica Acta, 1951;6:1-49.
4. Steele, B.T., Lireuman, D.S., and Beattie, C.W. Nephronophthisis. Amercican Journal of Medicine, 1980;68:531-8.
5. Straun, M.B. clinical and Patholoogical aspects of cystic disease of renal medulla. Annals of Internal Medicine 1962;57:373-81.
6. Fillartre, J.P. et al. Senior Loken Syndrome - a study of 8 cases from 5 families. Clinical Nephrology, 1976;5:14-19.
7. Manizer F, et al Familial nnephropathy associated with retinitis pigmentosa, cerebellar ataxia and sketetal abnormality. American journal of medicine, 1970;49:556-62...
8. Poopovic-Rolovic, M., et al. Juvenile nephronophthisis associated with rentinal pigmentary dystrophy, cerebellar ataxia and skeletal abnormalities Archives of disease in childhood 1976;51:801-3.
9. Boichis, H., et al. Congenital hepatic fibrosis and Nephronophthisis. Quarterly journal of medicine, 1973;42:221-33.
10. Prooesmans W.; Van Damme, B. and Macken J., Nephronophthisis and tapetaretinal degeneration associated with liver fibrosis. Clinical nephrology 3:160-4.

INFECTIVE ENDOCARDITIS PRESENTING AS SUBARACHNOID HEMORRHAGE

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Abstract: Involvement of the central nervous system during the course of endocarditis is uncommon and clinically important. The initial or presenting complaint involves the nervous system in a lesser number of patients. We report occurrence of subarachnoid hemorrhage, as the presentation of infective endocarditis of aortic valve.

Key words: Infective endocarditis, mycotic aneurysm, subarachnoid hemorrhage.

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Introduction:

A variety of neurological disturbances occur in about one-third of patients with endocarditis[1], including toxic confussional states, psychiatric symptoms and minor or major strokes, of which hemorrhage has been reported in 10 - 15% of cases[2]. We report a patient who presented with subarachnoid hemorhage (SAH) and was subsequently found to have infective endocarditis of aortic valve, an extremly rare clinical presentation.

Case History:

A 30 year old male was admitted in the emergency department, with two days history of high grade fever and severe global headache. On examination his temperature was 104°F, pulse rate of 112 beats per minute, bounding in character, blood pressure of 140/70 mm Hg in upper limbs and 190/60 in lower limbs, and other peripheral signs suggestive of aortic regurgitation. In addition signs of meningeal irritation were present, otherwise there was no apparent neurodeficit. Cardiovascular examination was suggestive of aortic incompetence. An impression of aortic value disease with possible endocarditis and meningitis was made. Investigations revealed hemoglobin of 9.7g/dl, TLC of 12800 with 76% polymorphs and ESR of 45mm/h (Westergren). ECG and chest x-ray revealed left ventricular hypertrophy. Urine examination showed 10-12 RBC/ high power field, routine biochemical investigations were normal, and three blood cultures grew viridans streptococcus. Echocardiography revealed aortic valuve regurgitation with multiple oscillating vegetations on the ventricular surface of aortic valve. Cerebrospinal fluid (CSF) was uniformly hemorrhagic and on examination, it showed 14000 crenated red blood cells /mm^3 and presence of xanthochromia. Cranial CT scan also showed blood in subarachnoid space. Revised diagnosis of rheumatic heart disease, severe aortic regurgitation with viridans streptococcal endocarditis and subarachnoid hemorrhage was made and patient was treated with antibiotics as per culture and sensitivity. His cardiac as well as neurological status improved and was discharged after four weeks of intravenous antibiotics in a stable condition and is under our regular follow up.

Discussion

Neurological manifestations occur in about one - third of patients with endocarditis including confusional states, psychiatric symptoms, meaningitis, brain abscess and strokes both due to embolism and hemorrhage[1,2,3]. Mycotic aneurysms develop in 2.5-15% of patients[1,2] and leakage from mycotic aneurysms occurs in less than 5% of patients and consequence of rupture of these aneurysms can be life threatening[2]. In these patients hemorrhage as a cause of stroke, can be a complication of either emboli or mycotic aneurysms[2]. Although patients with intracranial mycotic anneuryms are usually asymptomatic[4], severe unremitting headache can be a feature of intracranial aneurysm[5]. The diagnosis of ruptured intracranial aneurym is usually confirmed after neuroimaging studies of catastrophic headache[6], as in our case. The subarachnoid hemorrhage in our patient was most likely a consequence of mycotic aneurysm rupture. Various neurological manifestations as the presenting feature of infective endocarditis have been reported in 10-15 percent of cases, but subarachnoid hemorrhage as the presenting feature is an extremely rare presentation[2].

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Reference:

1. Korzeniowski OM. Kaye D. Infective Endocarditis: In Braunwald E (Ed.) Heart Disease - A Text-Book of Cardiovascular Medicine 4th Ed. Philadelphia, WB Saunders Company. 1992;1078-1105.
2. Sande MA, Kartalija M. Anderson J. Infective Endocarditis. In: Fuster V, Alexander RW, O'Rourke RA et al (Eds). Hurst's. The heart. 10th Ed. New York: McGraw Hill 2001;2087-2125.
3. Kaye D. Infective Endoscarditis. In: Fauci As, Braunwald E, Isselbacker KJ et al (eds.). Harrison's Principals of Internal Medicine 14th Ed. New York: McGraw Hill 1998;785-91.
4. Weinstein L: Life - threatening complications of infective endocarditis and their management. Arch Int. Med 1986;146:953-56.
5. Wilson WR, Giulliani ER, Danielson GK, Geraci JC. Management of complications of infective endocarditis. Mayo clin. Proc. 1982;57:162.
6. Cosmo LY, Risi G, Nelson S et al. Fatal hemoptysis in acute bacterial endocarditis. Am Rev Resp. Dis 1988;137:1223.

SYRINGOMYELIA WITH ATYPICAL PRESENTATION

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ABSTRACT: The classical presentation of syringomyelia is dissociated sensory loss, followed by motor weakness. We report a case of syringomyelia secondary to a high cervical cord tumour, with only motor symptoms and signs, which prompted us to report the same.

Key Words: Cervical cord tumours, syringomyelia, dissociate anesthesia.

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INTRODUCTION

Syringomyelia is a disorder characterized pathologically by the presence of long cavities in the central part of spinal cord[1,2,3]. It may be developmental as happens in the Chiari malformations, aquired from intramedullary spinal tumours, trauma or arachnoiditis. Occasional cases are idiopathic[1,3]. The usual features are weakness and atrophy of hands and arms, areflexia and dissociated type of anesthesia. Later on lower limbs may also get involved[2,3]. Unusual with our case is the absence of sensory loss and the occurrence of motor pathology and hyperreflexia only.

CASE REPORT

A 36 year old female, was admitted to the SMHS Hospital of Government Medical College, Srinagar with history of weakness of left upper limb, followed by involvement of left lower limb, then right lower limb and eventually weakness progressed to right upper limb. This weakness evolved insidiously over a period of one year, which progressed to the extent that patient became unable to walk even with support. There was no history suggestive of trauma to head or spine, nor was there any disturbance of sensation, bladder or bowel disturbance. The development of bed-ridden state brought the patient to seek medical attention. There was no such history of illness in the family.

On examination the patient was of overage built with visible wasting of small muscles of hands (left side more than that of right). Rest of the general physical, cardiovascular, and abdominal examination was normal. On neurological examination she was found to have normal higher functions, speech and cranial nerves. She was having wasting of small muscles of hands, which was more evident on left side. Tone was increased in both lower limbs. She had grade 4 power of upper limb muscles, whereas it was reduced to grade 3 on both lower limbs at hips, knee, ankles and feet. Her deep tendon reflexes were increased, in upper and lower limbs. Sustained ankle clonus was present bilaterally with upgoing planters. All the modalities of sensations were intact all over, after examining several times.

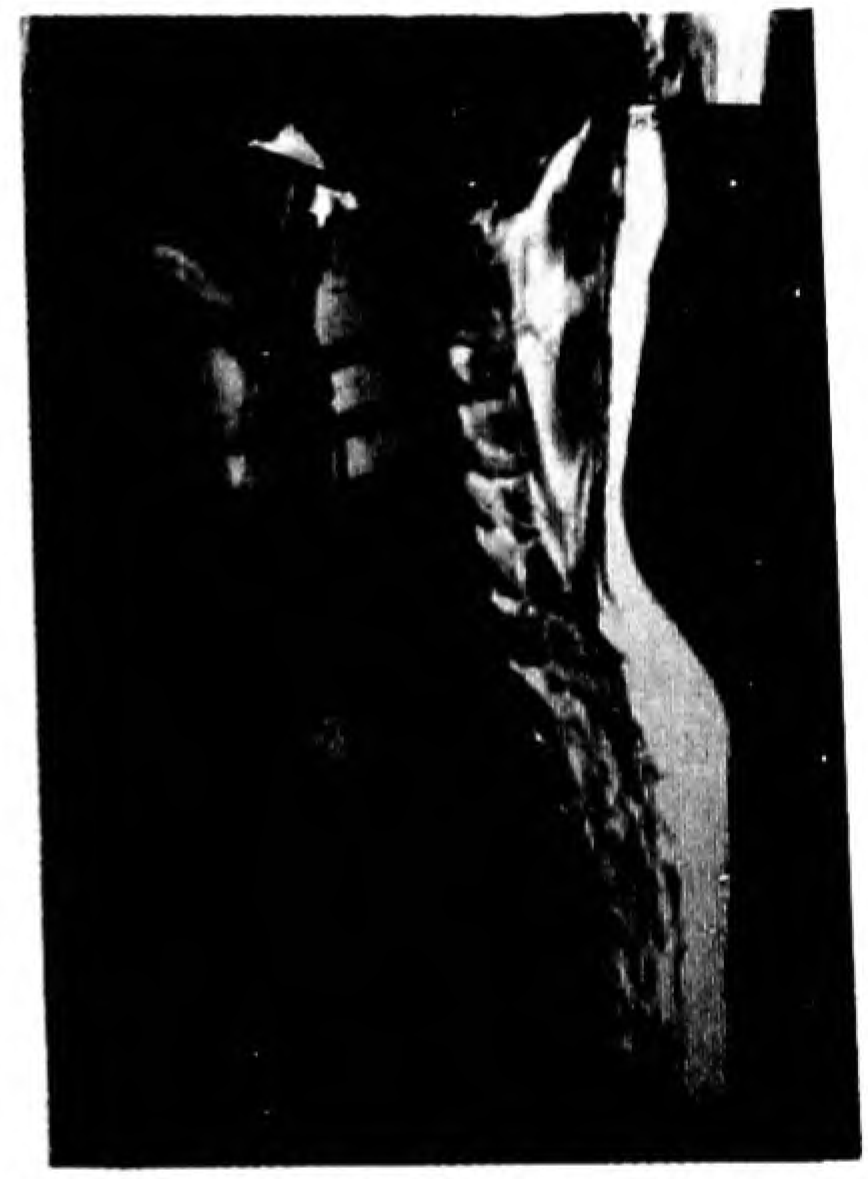

Fig. 1: Showing high cervical cord tumour with secondary syringomyelia.

All the routine investigations including biochemical parameters were normal, and radiological survey was not showing any abnormality, except MRI study (Fig 1) that revealed high cervical cord tumour (ependymoma) with secondary syrinx formation.

DISCUSSION

Usual clinical picture of syringomyelia in the high

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cervical cord comprises, painless weakness and wasting of hands and arms and segmental sensory loss of dissociated type (lose of temperature and pain sensation, and preservation of touch, joint position and vibratory sensations). Most cases begin with asymmetric unilateral sensory loss[1,2,3]. Finally weakness and ataxia of legs occurs due to involvement of corticospinal tracts and posterior columns in the cervical region[2,3]. Exceptionally, motor function is preserved, and the segmental dissociative sensory impairment may be the only marker of the disease process[2]. Reverse was true with our patient in whom there was no sensory loss but only the presence of motor involvement. It has been found very rarely in the literature, that no sensory loss be found in the presence of amyotrophy, and cases have been reported, in which only hydrocephalus and hydromyelia were present with spastic paraperesis[2]. Our patient had no evidence of hydrocephalus, but the mere presence of quadriparesis in association with syringomyelia secondary to high cervical tumour and absence of usual sensory loss, made it worth reporting, as the atypical presentation of syringomyelia.

REFERENCES

1. Hauser SL. Disease of the spinal cord. In: Fauci AS *et al* (eds). Harrison's Principles of Internal Medicine. 14th Ed. McGraw Hill, New York, 1988; 2381 – 90.
2. Victor M, Ropper AH. Diseases of the spinal cord. In: Adams and Victor's Principles of Neurology. 7th Ed. McGraw Hill, New York, 2001; 1293 – 1345.
3. Mc Lellan DL. Disorders of the spinal cord and cauda equina. In: Walton J (ed). Brain's Diseases of the Nervous system. 10th Ed. Oxford University Press, 1993; 478 – 512.

A CASE OF INFANTILE HYPERSEXUALITY - FREUD OR LEARNING

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ABSTRACT: *The phallic stage of psychosexual development is characterized by focus on the genitals around the age of 3-5 years. With the consequent development of the Oedipus complex, the primal scene may have been witnessed even prior to this life. We present the unusual case of a 21-month-old child who has developed a behavioral repertoire resembling sexual intercourse and with genital manifestations of sexual excitement. The role that primal scene witnessing and cognitive learning may play will be highlighted and their possible interactions discussed.*

Key word: *Infant hypers exuality, primal scene, cognitive learning.*

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INTRODUCTION:

According to Freudian Theory, beginning from the day a child is born and lasting till around 5 years, a child passes through certain welldefined psychosexual milestones. Freud believed, these are essential for normal sexual development in adult life. There has been ultrasound evidence that reflex erections occur in developing baby boys for several months in uteri, in the first minutes after birth and in baby girls there is vaginal lubrication and clitoral erection in the first 24 hours of life.[2] Kinsey reports that some times genital rubbing may lead to orgasm in infants less than 1 year old[3] Freud records in some detail the case of "little Hans" a 5 years old, who had phobia for horses and an excessive interest in genitalia and the sensations he derived from them.[4] The development and resolution of the Oedipus complex are essential to the role of the ego as an organizing unifying agent of one's past and the development of the superego. A biological time basis for the Oedipus complex may be indicated by observations of human pre-historic and non-human primate behavior.[5] A peaking of human infantile sexuality at age of 5 or 6 years may be the vestige of an earlier biological timetable. Till date role of learning has not been elucidated in the development of human sexual behavior; cognitive learning in particular may have a role to play as will be illustrated below:

CASE REPORT:

A 21 month old male child was referred to the OPD from a private clinic with a one month history of unusual behavior. Whenever the child noticed anyone lying down in the house, he would pull up her or his shirt, stand against the back or abdomen with a crunched stance and start a backward and forward thrusting motion, rubbing his penis against the naked skin, accompanied by frequent sighs of pleasure and occasional shouts. If allowed to go on, he would continue for period of up to thirty minutes after which he would start sweating profusely, sit-down and often start crying and banging his head against the wall. The parents report that during this act, the child's penis was erect and detumescence would occur only after he had stopped. This would occur on almost a daily basis and some times even 2 to 3 time a day. The parents reported that this peculiar behavior would seemingly begin after the child's paternal uncle's wife would jokingly ask him to message her tummy and he would take great delight in doing so. The child also repeated this behavior most often with her, particularly as she would not stop him as she felt that it was natural for a child. The child's father however was very upset by this and felt there was something seriously wrong with him. He had even contemplated killing the child as he felt that this action was similar to that of the Devil. There was a great reluctance to disclose further information about the rest of the family. The child's family is an extended one, living in a rural area with the child's parents, two siblings and the paternal uncle and his wife who are issueless. The child's divorced paternal aunt and her two teenage daughters also lived in the same house. The older brother of the child has a history of frequent temper tantrums and nocturnal enuresis. The family is cohesive with low levels of stress. Overindulgence of the child by paternal uncle and his wife occurs frequently and he often sleeps with them. The child was a FTND at home with no significant antenatal or perinatal problems. Developmental milestones were consistent with the age of the child.

Temperamentally, the child is very sociable and adapts readily although he tends to be aggressive when thwarted.

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Since the age of 1 year, infrequent autistic behaviors have been noticed including head holding, persistent staring at objects, head banging, rocking and repetition of syllables. None of these were apparent during observation. On observation the child was well nourished and groomed. Attention and concentration were normal, activity levels were within normal limits, motor behavior included an ability to copy straight lines. The child combines two words readily and learns new words with just 2-3 repetitions. He remains absorbed in play, engages in symbolic play and recognizes classes of objects, displays affection frequently. On prompting by the father who sat down on the floor, the child pulled up the father's shirt and started the behavior described previously. He continued until distracted. Physical examination including hearing, vision, head circumference, height, weight, external genitalia, was unremarkable. Systemic examination was within normal limits.

Investigations included an x-ray skull showing a normal sella and C. T. brain, which was normal. Liver function, Blood counts, Kidney functions, Sr. Calcium were within normal limits. An EEG done was appropriate for the age of the child. The child was started on a course of aversion therapy in the form of conditioned negative reinforces coupled with positive reinforcement. The parents were counselled and reassured. Over 4 sessions, the frequency of the child's behavior had reduced significantly. Environmental modifications in the form of reducing overindulgence, reduced control of disciplining by the uncle's wife were also attempted but with strong resistance to change from the family.

DISCUSSION:

The development of such an elaborate repertoire of sexual behavior in a 21 months old child is extremely unusual. There has obviously not been enough time for the full process of psychosexual development to take place. Even the roles that aggressions, object relations, ego and superego functioning or sense of self could play are extremely limited. One of the youngest children described is "little Hans" a 5-yearold boy with a phobia of horses and an extremely early interest in sexuality starting around the age of 31/2 years.[4] The analysis of the Child's history and his treatment as described by father, Freud opened up new avenues in psychoanalysis and is considered a prelude to infantile analysis. But "little Hans" had reached a much higher level of cognitive and language development, being able to reason and test between reality and fantasy. He had reached the stage of categorization in language development. In contrast, the baby under discussion was just at the modification stage of his language development and cognitively was still in the sensor- motor stage. Where then does such behavior arise? Naisberg claims that early homeostatic disruption impairs the Inherited Schemata Representation (ISR) program for gender definition and sexual orientation with postnatal asexuality being replaced by 'Infantile Sexuality' operating on an impulse like pattern.[6] However to establish the presence of disruption as well, such schemata are currently not practicable and further research in the area has been fruitless. Witnessing of the primal scene or the parents engaging in sexual intercourse was described by Freud in the case of the "Wolf man"- a case of infantile neurosis with an animal phobia and the later development of an observational neurosis.[7] The patient witnessed the act apparently, at the age of 11/2 years and Freud comments that such an occurrence must be commonplace in life. Konen and Rechard[8] comment on the fact that primal scene phantasies reflect the shaping of oedipal sexuality throughout life and are almost universal.

In the case of baby under discussion, such a possibility was vehemently denied by the parents but it is entirely possible that childless paternal uncle and his wife could have let such an event happen.

Cognitive learning from observation and as a part of conditioned learning has been described even in chimpanzees.[9] Here; instead of stimulus - response associations, stimulus-stimulus association is learned. We hypothesize that in case of the baby under discussion, the witnessing of the primal scene resulted in cognitive learning and the subsequent development of the behavioral repertoire described above. The above average intellectual capability of the infant would be a fertile substrate for such a happening.

Further investigation into the role that learning plays in psychosexual development are indicated. The quite uncommon case of the baby under discussion, gives us a singularly unique insight into how sexuality may develop in infancy.

REFERENCES:

1. Freud S. Three Essays on the theory of sexuality. Standard edition of the complete psychological works of Sigmund Freud. James Strachey (Ed.) Hogarth Press. London 1953: 7, P. 123-245.
2. Masters WH, Johnson VE, Kolodny RC: on Sex and human loving childhood sexuality P-124 Jaico. New Delhi 1999.
3 Kinsey AC, Pemeroy WB, Martin CE, Sexual Behavior in the Human Male. Philadephia, Sundees, 1948.
4. Freud S. Analysis of a Phobia in a five year old child. Standard edition of the complete psychological works of Sigmund Freud. James Strachey (Ed.). Hagarth Press. London 1953, 10. P 1-149.
5. Jonas AD, Jonas DF; A Biological basis for the Oedipus complex, an evolutionary and ethological approach. Am. J. Psychiatry 1975.Vol -132, page no. 602
6. Naisberg 4; Homestatic disruption and sexual disorders. Med Hypotheses 1996; 47: 243-7.
7. Freud S; from the history of an infantile neurosis. Standard edition of the complete psychological works of Sigmund Freud. James Strachey (Ed) Hogarth press London 1953; 91-122.
8. Ikonen P, Rechuedt E, on the universal nature of primal scene phantasies, Int. J. Psychoanal 1984 65:- 63-72.
9. Morgan CT, Introduction to Psychology King RA, Weisz JR, Schopler JS (Eds.). Principles of learning 1993 Pg. 168. Tata McGraw Hill, New Delhi.

LOCALIZING PARATHYROID ADENOMA WITH TECHNETIUM-99M SESTAMIBI RADIONUCLIDE SCAN

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ABSTRACT: We present a case report, documenting the utility of radionuclide scintigraphy in a patient with parathyroid adenoma. This method is very sensitive in confirming the diagnosis and accurately localizing the site of parathyroid adenoma.

Key words : Parathyroid adenoma, Radionuclide scintigraphy, Tc-99m Sestamibi

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INTRODUCTION

Primary hyperparathyroidism affects one of every 1000 persons. One in every 500 women over age 45, and one in every 2000 men.(1) 80% of cases of primary hyperparathyroidism are due to solitary parathyroid adenoma.Between 10-15% of cases are due to hyperplasia of more than one gland, commonly all four. Pre-operative localization of a parathyroid tissue may allow a reduction in operation time, unilateral neck exploration or key hole/ thoroscopic surgery. A variety of imaging techniques have been developed to accurately localize the abnormal gland (s) prior to surgery and over the last three decades a number of methods have been used to localize parathyroid glands with radionuclides. Initially radiocynacobalmin (Co-Vitamin D12) and Se-selenomethionine were used with varying success.(2,3) Nowadays Thallium Scanning or Technetium-99m sestamibi scanning is used with varying degree of success to localize the parathyroid gland. We present a case of parathyroid adenoma with derranged Sr. calcium and PTH levels showing an increased persistent tracer uptake of Tc-99m sestamibi in the neck at varying intervals of time below the right lobe of thyroid gland. Similarly Tc-99m MDP bone scan revealed intense metabolic activity in metaphysis of both femurs, tibias and humerus signifying the bone involvement also.

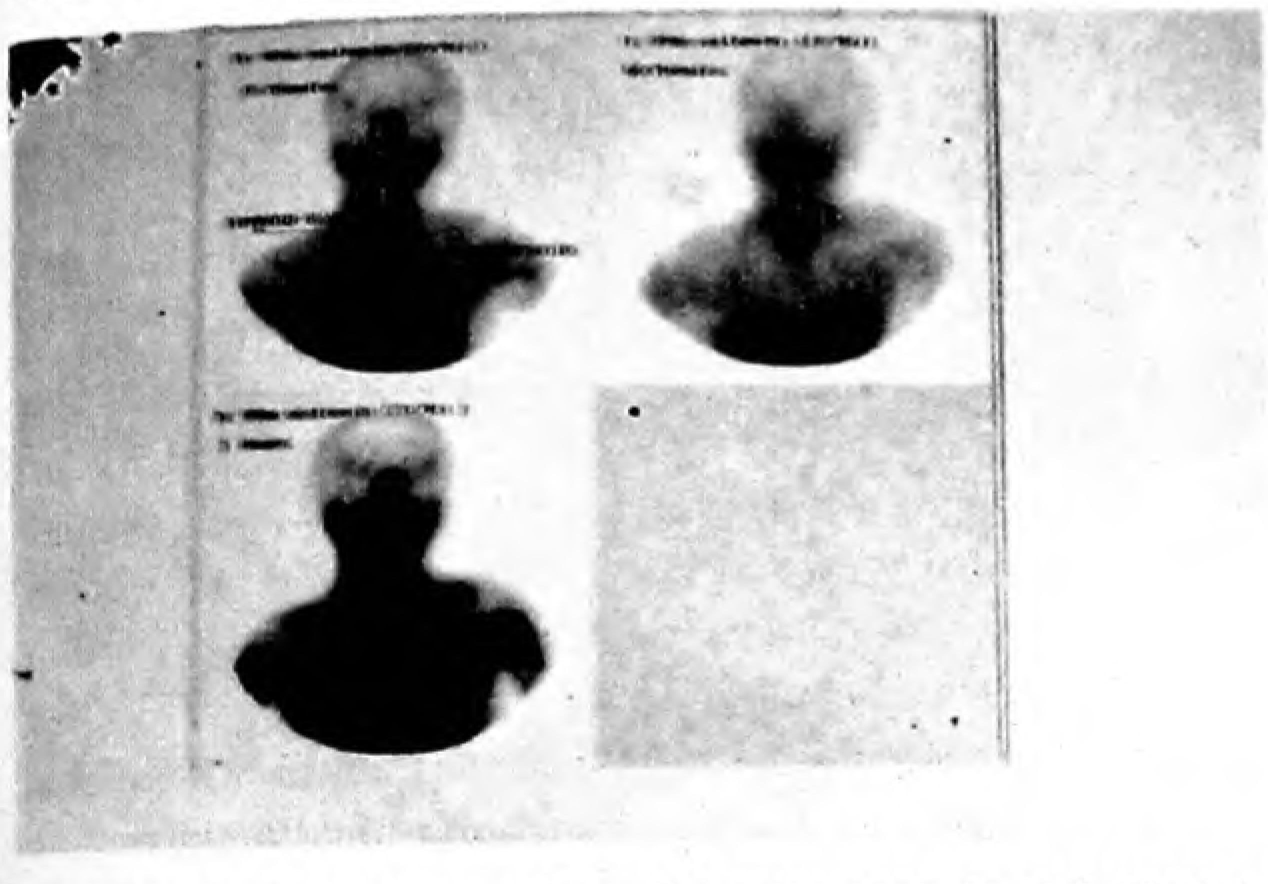

Figure 1: Shows parathyroid adenoma in relation to the lower pole right lobe with concentration of Tc-99m sestamibi which persists at 60 minutes, and 3 hours, suggestive of parathyroid adenoma.

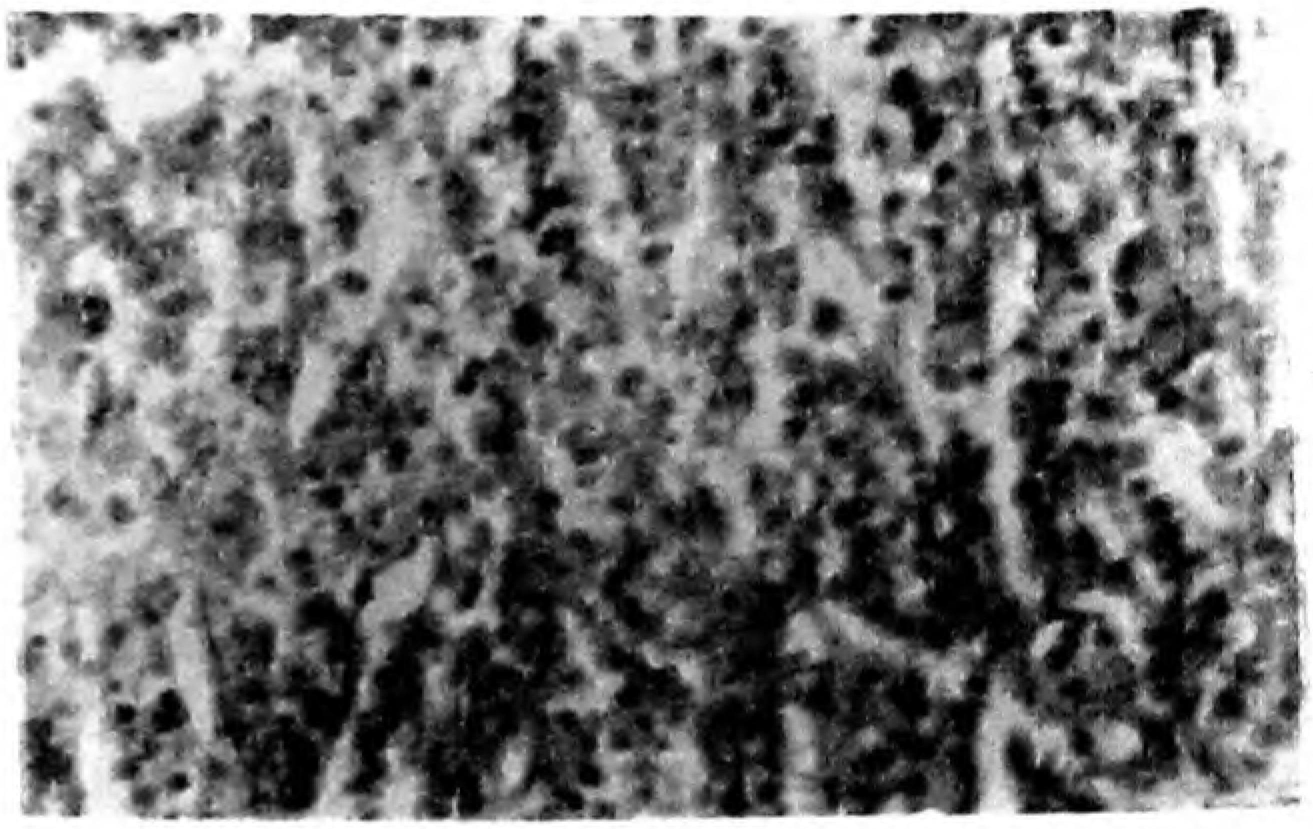

Figure 2: Shows parathyroid aenoma of this patient seen under high power microscopic x 400 H and E stain.

CASE REPORT

A 16 year old male presented with history of non-traumatic fracture left femur 1 1/2 month duration, weight loss, constipation and depression. General physical examination and system examination were normal. Clinical

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SENSTIVITIES OF IMAGING MODALITIES IN LOCALIZING PARA-THYROID TUMOR.

		Adenoma %	Hyper-plasia %	Ectopic % Med. Region	Re-operation %
1.	Scintigraphy				
	T1-201/Tc-99m	35-92	47-80	80-86	26-70
	RTB/Tc-99m	86-93	63-97	90-100	100
2.	Ultrasonography (High resolution)	34-92	53-78	0	36-82
3.	CT (High resolution & contrast enhancement)	41-86	0-54	47-70	47-63
4.	MRI	57-90	0-75	50	50-78
5.	Angiography	77	NA	NA	54-85
6.	Venous PTH sampling	75-90	NA	NA	69-80

RTB = Radioactive toluidine blue; Tc = Technetium; T1 = Thallium; CT = Computed Tomography; MRI = Magnetic Resonance Imaging; PTH = Parathyroid hormone

and Biochemical investigations showed evidence of hypercalcemia 16.1 mg/dl (Ref. range 8.5-10.1) and increased levels of PTH 345H (Ref 12.0 - 72.0). Rest of the parameters were normal. A total body bone density scan revealed gross severe osteoporosis with BMD 0.652 g/m^2

A Tc-99m sestamibi parathyroid scan was performed and sequential images were taken at 15 minutes, 60 minutes and 3 hours. A marker was placed on the sternal notch to precisely localize the parathyroid gland. The scan showed persistent focus of tracer uptake below right lobe of thyroid with complete washout of the tracer from the thyroid glands in the 60 minute and 3 hour images. The washout fraction calculated for parathyroid gland between 15 minutes and 1 hour was 7.3% and between 15 minutes to 3 hours was 61.2%. Similarly the washout fraction calculated for thyroid gland between 15 minutes and 1 hour was 34.3%. A Tc-99m MDP whole body bone scan was done 3 days later which revealed instence tracer uptake at the lower metaphysis of both femurs, upper metaphysis of both tibia and upper metaphysis of both humerus suggestive of intense metabolic activity.

DISCUSSION

A number of imaging modalities are available for localization of parathyroids and include both invasive and non-invasive methods. They are:

Invasive

- ☐ Arteriography
- ☐ Digital subtraction angiography
- ☐ Selective parathyroid venography
- ☐ Imaging procedures to guide parathyroid aspiration biopsy.

Non-invasive

- ☐ Ultrasonography
- ☐ Radionuclide scintigraphy
- ☐ Computed tomography
- ☐ Magnetic Resonance Imaging

Radio-nuclide imaging

The ideal radio-pharmaceutical for parathyroid imaging should not only have favourable imaging characteristics and dosimetry but also a high sensitivity and specificity for parathyroid tissue. A number of agents have been used in the past but with limited success. Recently ^{201}T1/Tc-99m based subtraction method has been used frequently on the fact that ^{201}T1 is taken up by thyroid and parathyroid tissues whereas Tc-99m pertechnetate is only taken up by the thyroid. However, there are several limitations to this method. They are:

1. Tracer uptake is related to distribution of potassium. It may occur in overlying thyroid tissue and in thyroid abnormalities.
2. Low turmoral uptake limits detectability of small hyperplastic glands resulting in 55% sensitivity.
3. Artifacts may occur as a result of motion during imaging.
4. Locating a parathyroid adenoma in blind spots such as mediastinum with ^{201}T1 scintigraphy may be difficult.

As a result of above limitations, recently Tc-99m sestamibi has been used as an alternative to ^{201}T1 for parathyroid localizations.[4,5] Tc-99m sestamibi is a cationic complex which has a different biokinetic handling in thyroid and parathyroid tissue,(5,6) enabling the possibility of single agent use if delayed imaging is performed. The difference being due to number of mitochondria in cells because adenomas have a large number.(7) The sensitivity of Tc-99m sestamibi scanning in detecting both adenomas and hyperplastic parathyroid glands is higher with sestamibi than with thallium. Moreover, superior imaging characteristics, improved dosimetry and a better target to

LAURENCE – MOON (BARDET) BIEDL SYNDROME

Tariq Qureshi; Ayub; Prof. A.R.Nasti; Mehmooda Ashai

Abstract: We report a case of Laurence Moon Biedl syndrome with Pendular nystagmus as the uncommon feature in addition to the five cardinal features.

Key words: Retinopathy, Polybactyly
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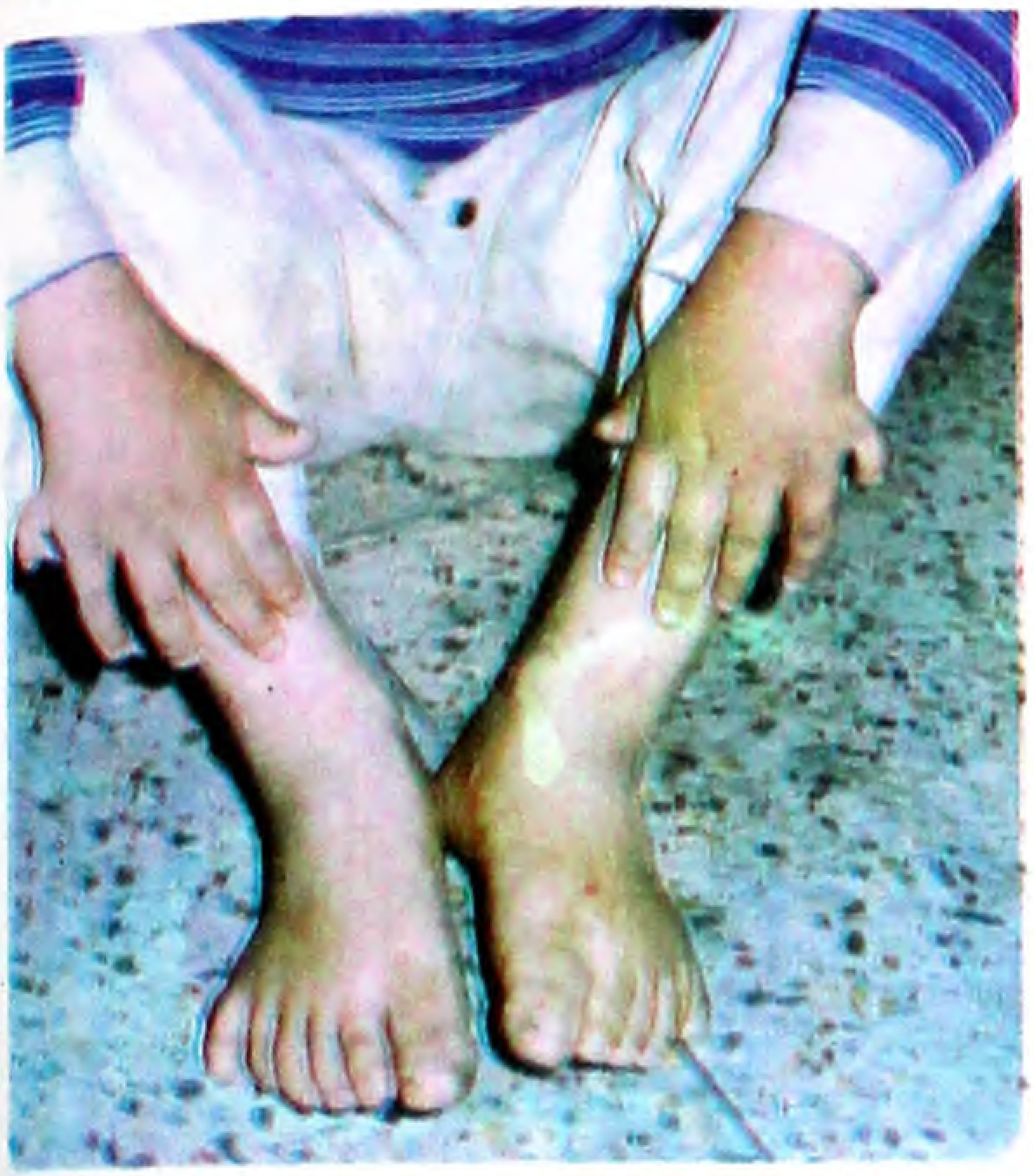
INTRODUCTION

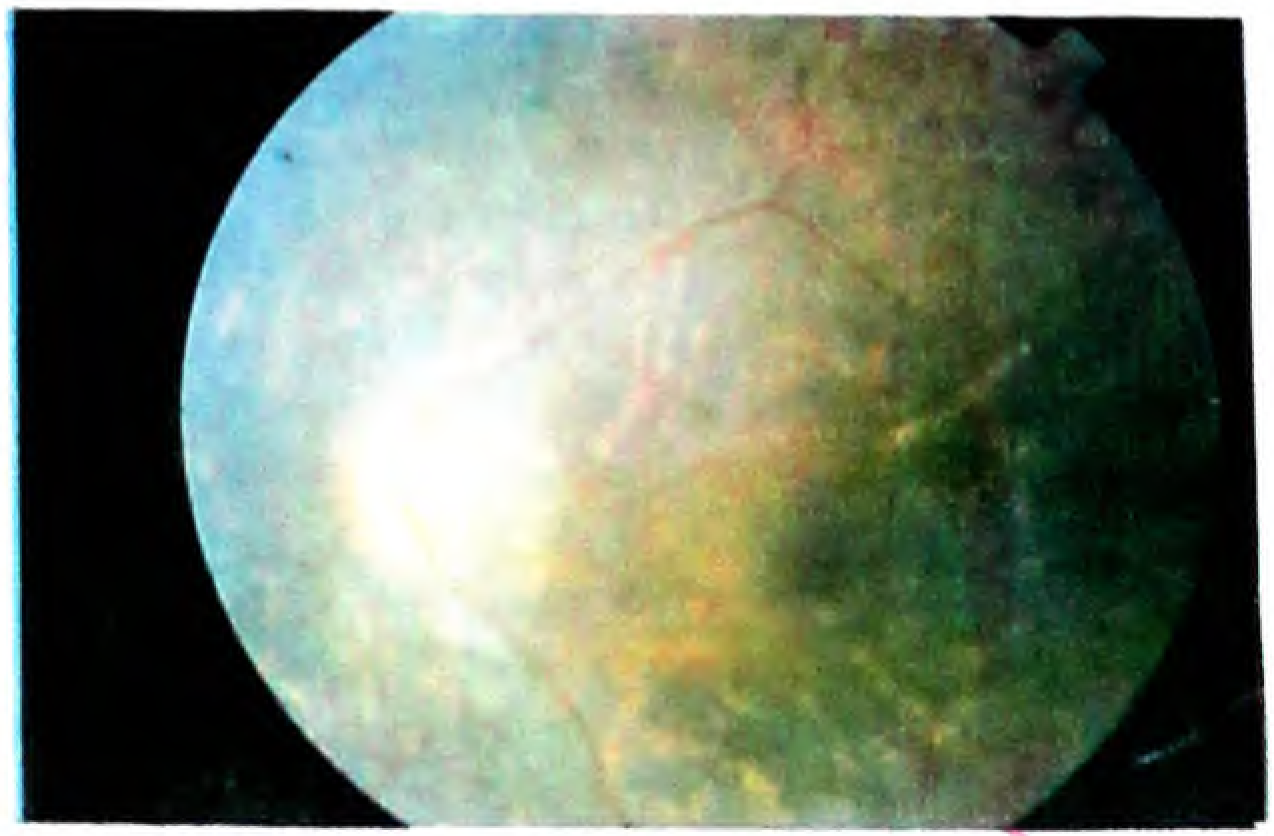
Laurence-Moon (Bardet) Biedl Syndrome, first defined by Bardet in 1922 [1] is an autosomal Recessive disorder characterized by structural and functional abnormalities of organs and tissues with diverse embryonic derivation.

The five cardinal features of the syndrome include Polydactyly or Syndactyly, Pigmentary retinopathy, obeisty, Mental retardation and Hypogonadism [2]. Other systemic features include Brachycephaly, short stature, congenital Heart Block, Deafness, Neurological disorders and Kidney disorders [2,3,4].



Patient generally has onset of symptoms within the first 10 years of life and most often the first complaint is poor night vision [5]. Nystagmus is a common feature. Peripheral visual fields are constricted. Fundus changes include constricted arterioles, waxy disc pallor and peripheral pigmentary changes [5] including pigment atrophy, Bone specular pigmentation and areas of white deposits.

Unlike classic Retinitis pigmentosa unassociated with systemic disease, these patients show degenerative changes within the macula at an early stage with relentless earlier decrease in the central vision and most patients are legally blind by age 30 ERG shows diffuse photoreceptor disease[5].



CASE REPORT

A 10 year old boy reported to eye O.P.D. with complaints of poor night vision and Jerky eye movements of uncertain duration and onset, and of progressive nature. There was no history of consanguinous marriage between parents, complicated vaginal delivery or any intra-uterine infection. The other male sibling who was 2 years elder and the parents were a-symptomatic.

Systemic examination revealed polydactyly (Hexadactyly), obeisty of truncal type also involving proximal extremities, Mental retardation (I.Q <60), Hypogonadism (Pea nut size tests) and sensoneural hearing loss. Cardiovascular and urinary system were normal.

Ophthalmic examination revealed orthophoria, normal ocular

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movements and pendular nystagmus in both eyes.

The unaided visual acuity in both eyes was 6/60 correctable to 6/12 with –1.50D.sph. and –0.50 D.cyl. Axis 90°. Slit lamp examination of the anterior segment was normal.

Fundus examination revealed constricted arterioles, waxy disc pallor, Peripheral pigmentary changes including areas of white deposits and bone spicule pigmentation. The foveal reflex was dull in appearance in both eyes.

Lab. Investigations including CBC, ESR, KFT and Urine examination were normal. Chest and abdominal Roentgenogram were normal. Serological tests were unrewarding. Abdominal ultrasound and Echocardiogram were also normal.

DISCUSSION

Laurence – Moon (Bardet) Biedl syndrome is a rare genetic disorder first defined by Bardet in 1920 [1]. He described three of the five cardinal features of the syndrome i.e; Polydactyly, Obeisty and Pigmentary retinopathy.

Biedl in 1922 [1] added mental deficiency and genital hypoplasia to this syndrome. In 1925 Solis-Cohen and Weiss [6] connected to this syndrome the four patients in one family described by Laurence and Moon, in 1966.

Solis – Cohen and Weiss coined the name Laurence Moon Biedl syndrome. The cases reported by Laurence and Moon were re-evaluated and reported by Hutchinson [7], the members were found to have a disease characterized by typical pigmentary retinopathy, Mental retardation, arrest of sexual development and progressive weakness leading to paraplegia.

Confusion exists in medical literature regarding the differences between Laurence Moon and Bardet Biedle syndrome. Common to both are pigmentary retinal degeneration, Mental retardation and Hypogonadism. Spastic paraplegia is predominant feature in Laurence Moon syndrome, Polydactyly and Obeisty are predominant in Bardet Biedl syndrome. Some researchers believe that Bardet Biedl Syndrome is a subdivision of Laurence Moon syndrome. Hence the term Laurence Moon Biedle syndrome has gained universal acceptance in the world literature.

CONCLUSION

This case illustrates several common features of Laurence Moon Bardet Biedl syndrome including the five cardinal features i.e; Polydactyly, Obeisty, Mental retardation, Hypogonadism and Pigmentary retinopathy.

In addition the patient has pendular nystagmus and sensorineural hearing loss which have been reported less frequently in other series.

Reference:

1. Mc Louglin TG, Shanklin DR. Pathology of LMBBS. J.Pathol 1967; 93: 65-78
2. Yamada K, Miura M, Miyayama H, Sakashita N, Kochi M, Ushio Y Diffuse Brain Stem glioma in a patient with LM(B)B syndrome. Pediatr Neurosurg 2000 Dec; 33 (6): 327-7
3. Urben SL, Baugh RF. Otolaryngologic features of LM(B)B syndrome Otolaryngol Hend Neck Surg. 1999 April; 120 (4): 571-4
4. Ukar B, Yakut K, Kural N, Buyukasik F, Vardareli E Renal involvement in LM(B)B syndrome. Pediatr Nephrol 1997 Feb; 11(1): 31-5
5. Riise R, Andreasson S, Wright AF, Tornqvist K. Ocular findings in LM(B)B syndrome Acta Ophthalmol Scand 1996 Dec; 74(6):612-7
6. Solis – Cohens, Weiss E The Laurence Biedl syndrome: a report of four cases in one family Am J Med Sci; 1925; 169: 489
7. Hutchihson J. Slowly progressive paraplegia and disease of choroid with defective intellect and arrested sexual development in several brothers and a sister. Arch Surg (Lond) 1900; 2: 118.
8. Laurence JZ, Moon RC. Four cases of R.P. occuring in the same family and accompanied by general imperfections of development Ophthalmol Rev 2:32, 1886.

background (parathyroid : thyroid) ratio at different time points superceds the use of ^{201}T1 for parathyroid imaging and the ease of a single agent method use of Tc-99m sestamibi is likely to increase further in future. In the present study the above mentioned advantages are very clearly illustrated. As a matter of routine, convenience and technical ease, all the patients with clinical and biochemical evidence of hyperparathyroidism should be subjected to a pre-operative Tc-99m sestamibi imaging. This not only helps in confirming the diagnosis but also in localizing the site of adenoma thus facilitating surgical exploration.

REFERENCES

1. S Tzila Zwas, Abrahain Czerwak. The parathyroids. Principles of Nuclear Medicine. By Wagner, Szabo, Buchannan 2nd edition 1995; 639-651.
2. Ell PJ, Tood-Poksopek A, Britton KE. Localization of parathyroid adenomas by computer-assisted parathyroid scanning. Br J Surgery 1975; 62: 553-55.
3. Robinson RJ. Parathyroid scintigraphy revisited. Clin Radio 1982; 33: 37-41.
4. Coakley AJ, Kettle AG, Wells CP. Tc99m Sestamibi - a new agent for parathyroid imaging. Nuclear Med Commun 1989; 10: 791.
5. O'Doherty MK, Kettle AG, Wells P, Collmas RE. Parathyroid imaging with technetium- 99m-sestamibi. Pre-operative localization and tissue uptake studied. J Nucl Med 1992; 33: 313.
6. Tallefer R, Boucher Y, Potviuc C. Detection and localization of parathyroid adenomas in patients with hyperparathyroidism using a single radionuclide imaging procedure with ^{99}Tc-m-sestamibi. J Nucl Med 1992; 33: 1801-07.
7. Sandrock D, Merino MJ, Norton JA. Ultrastructural histology correlates to thallium2201/technetium-99m parathyroid subtraction scintigraphy. J Nuclear Med 1993; 34: 24-29.

PANCYTOPENIA IN A BRUCELLOSIS CASE INVESTIGATED FOR PYREXIA OF UNKNOWN ORIGIN

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Abstract: *We report a case of brucellosis in a young, Gujjar, woman with family occupation of sheep and cattle raising, admitted as a case of fever of undetermined origin with anemia, who showed pancytopenia as a prominent feature.*

Key words: Brucellosis, Fever of unknown origin, Cattle rearing, Pancytopenia.

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Introduction:

Brucellosis, a globally prevalent major zoonotic disease, is notorious for its atypical presentations often making the diagnosis difficult. Patients may be asymptomatic; present with a typical bout of acute brucellosis; have a short episode of trivial illness; have atypical, misleading presentations as flu-like syndrome, arthralgias, vague aches and pains, different types of rashes etc; or present in its protracted form with chronic fatigue, malaise, low backache, psychosomatic disorders, etc. In countries where the condition is endemic, as the Mediterranean, Middle East, Mexico, South Africa, and parts of the USA (1-4) with strong suspicion among the practitioners, diagnosis is early, but in areas where disease occurs sporadically, diagnosis is generally delayed causing great suffering, mental anguish and financial losses. In the State of Jammu & Kashmir, where rearing of cattle is common practice among villagers and fever of unknown origin not infrequent, reporting of brucellosis is rare.

Case history:

A 22 year old female of Gujjar ethnicity, a nomadic tribe famous for traditional cattle & sheep breeding practice, reported to the medical out-patient department with chief complaints of fever, weakness, burning micturition and generalized aches and pains of 20 days duration and dry cough for the preceding 8 days. Fever was higher and accompanied with chills in the mornings, and there was no history of any evening rise of temperature or sweating. Additional symptoms included palpitations, breathlessness and easy fatiguability.

On examination, the patient was febrile and anemic. Auscultation of chest revealed bilateral crepts; both the heart sounds were heard with a systolic murmur. Abdominal palpation revealed hepato-splenomegaly. Examination of the nervous system

Revealed no positive findings. Results of laboratory investigations are given in the box.

CBC:

1. Hemoglobin 4.5 g/dl
2. WBC: 1,300 /cu. mm.
3. RBC: 2.78 million /cu.mm.
4. Platelets: 83,000 /cu.mm.
5. DLC: Neutrophils 35%; Lymphocytes 60%; Others 5%

Urine: Albumin: Traces; Sugar: absent; WBC: 2-3 /HPF, with few epithelial cells

Blood chemistry:Blood Urea, 32 mg/dl
Serum creatinine, 1.0 mg/dl
Sugar (fasting), 68 mg/dl
Serum sodium132 mEq/L serum potassium 3.2 mEq/L

Liver function:	Normal
Bone marrow:	Dual deficiency anemia, with inflammation
USG:	Hepatosplenomegaly with minimal collection in pouch of Douglas.
X-ray:	Lung fields clear with normal heart size
ECG:	Within normal limits

Widal's Agglutination test: Negative

Wright's agglutination tests: Positive titres, exceeding 1:1,024, for both *B. abortus* and *B. melitensis.*

Discussion:

In the Jammu & Kashmir State, which is endemic for enteric fevers, pyrexia of unknown origin is generally attributed to typhoid fever, with practitioners, registered as well as unregistered, resorting to a long empirical therapy of some currently fashionable antibiotic before embarking on relevant investigations. A very low grade suspicion of

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presence of brucellosis, due to its generally asymptomatic nature or atypical presentation, compounds the under-diagnosis, with the result that the patient suffers for a long time before the correct diagnosis is made and the patient rid of his infection. Widal's test is almost routinely done in all cases of prolonged fever but awareness about the Wright's test is limited among the rural practitioners, or else the test is not available in most of the rural laboratories. Cultures, which anyway take not less than 4 weeks, are available only in the tertiary care hospitals in urban areas. Although no single test other than isolation of brucellae from the patient can be relied upon with certainty (5), diagnosis of brucellosis in rural areas of our State has to be based on some suggestive investigations which are possible to perform in local low-cost, low-tech, laboratories of the primary health care delivery system.

Getting a complete blood count done is practicable at all levels of rural health care, and leads to least patient or familial inconvenience, since the film can be collected at patients' home even in remote areas. Pancytopenia has been seen consistently in patients of brucellosis with diverse presentations (6-9). Single stem-cell dysfunction such as anemia, leucopenia or thrombocytopenia is also common. (6-10). Granuloma in bone marrow can be seen as an evidence of haemophagocytosis (6, 10). Our patient had pancytopenia with inflammatory evidence in the bone marrow.

Conclusion:

In rural India, where facilities for serologic diagnosis of brucellosis may not be always present, simple blood counts could point to the presence of brucellosis and help the practitioner to arrive at any early diagnosis or at least to consider brucellosis in the differential diagnosis of pyrexia of unknown origin.

References:

1. Kadri SM, Rukhsana A, Laharwal MA, Tanvir M. Seroprevalence of Brucellosis in Kashmir (India) among patients with pyrexia of unknown origin. JIMA 2000;4:170-1
2. Anil Sachdev, Rakesh Vohara, Sunita Bijaria. Acute brucellosis of child: A case report with unusual features. Indian Pediatrics 2001;38:1421-1425
3. Young EJ An overview of human brucellosis. Clin Infect Dis 1995; 21:283-290
4. Texas Department of Health: Under reporting of four infectious diseases by Texas hospitals. Dis Prev News 1993;53:1-3
5. Wohl MG Brucellosis. Internal Medicine Its theory &Practice Lea & Febriger, Philadelphia 1951;182-190
6. Crosby E, Llosa L, Quesda MM et al. Brucellosis as a cause of severe colitis. Am. J Gastroentrol 1991;86:917-919
7. Stermer E, Nissim L, Potasman I et al. brucellosis as a cause of severe colitis. Am. J Gastroentrol 1991;86:917-919
8. Ila R, Young E. Acute Brucella ileitis. Am. J Gastroentrol 1988;83:80-82
9. Swenson RM, Cundy KR. Human infection with Brucella canis. Ann. Intern Med 1972;76:435-438
10. Schussler JM, Fenves AZ, Sutker WL.Intermittent fever and pancytopenia in a young Mexican man. South Med J 1997;90(10):1037-9

CA-125 TUMOUR MARKER FOR OVARIAN MALGNANCIES

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Of all the gynaecologic cancers ovarian malignance represent the greatest clinical challenge because they are usually asymptomatic until they have metastatised,patients present with advanced disease in more than 2/3rd of the cases .Epithetial cancers are the most common ovarian malignancies and has the highest fatality to case ratio of all the gynnaecologic malignancies (1).Ovarian cancer is the sixth most common cancer in the united states accounting for 4% of all the female cancers and 25% of the cancers of the female genital organs.a women having risk of having ovarian cancer in her life is nearly 1.5%.so there exists a need for tumor marker, which help in screening, early diagnosis,staging,assement of treatment response and follow up.The various tumor markers tested for ovarian carcinoma are CA-125,CEA,CA 19.9,CA 72.4, placental alkaline phosphates (PLAP),cancer associated serum antigen (CASA),CYFRA 21.1,OVXI,TPA etc.At present serum CA-125 is the gold standard tumor marker in the evolution of pelvic masses,particularly ,in ovarian epithelial cancer.A breakthrough occured in 1983, when Bast and associates reported on an immunoradiometric assay using a monoclonal antibody that recognised the Mullerian derived glycoprotien antigen designated CA-125.This marker is elevated in more than 80% of non-mucinous ovarian epithelal cancers,30% of non ovarian malignancies,6%of benogn gynaecologic disorders and only 1% of normal cases. The majority of benign conditions that cause elevation of CA-125 include pregnancy, ovarian cysts, uterine leiomyomas, pelvic inflammatory disease,endometriosis and menstruation.CA-125 is a mucin like, high molecular weight (200-1000 KDa) substance.Half-life in body is usually 20 days. Blood specimen for CA-125 should always be taken when a female is not actively menstruating. Upper limit for a normal serum level of CA-125 is commonly 35 IU/ml and above this level is suspicioun of ovarian malignancy. Markedly elevated CA-125 levels correspond with stage and tumor bulk in FIGO at stage II through IV.The serum CA-125 concentration is >35 IU/ml in about 80% of women with carcinoma‘ ovary and 26% of women begin ovarian tumors. Only 3% and 0.8% of normal healthy women, have a CA-125 concentration over 35 and 65 IU/ml, respectively(2).An ideal tumor marker should have 100% sensitivity, specificity and positive predicitive value. The senitiviy of CA-125 estimation for ovarian carcinoma of epthelions is 69.8%,specificity 72.3%, positive predictive value 49.2% and negative predicitive value 86.2%.

Screening

Consider the magnitude of malignancy,the international community felt strongly about the requirement of screening markers. A combination of two or three markers was not superior to single marker for screening[3]. At present because of false positivity, particularly in premenopausal women, CA-125 is not cost effective and should not be used routinely to screen carcinoma ovary[4]. However it is recommended that high risk group (i.e. women with family history of one or more first degree relatives having ovarian carcinoma in postmenopausal age, presenting with a pelvic mass) should be selected for the screening by CA-125 levels.

Diagnosis and Staging

Serum CA-125 is the current gold standard tumor marker in the evaluation of pelvic masses.To distinguish benign conditions from malignant standard cut off point of 35 IU/ml,can be misleading especially in menstruating women.CA-125 cut off point from 65 to 200 IU/ml is necessary to distinguish benign conditions from malignant(5).Fifty percent of stage I/II patients show elevated levels of CA-125. Markedly elevated CA-125 levels correspond with stage and tumor bulk in FIGO stage II through IV of ovarian carcinoma (4). In postmenopausal women with an asymptomatic pelvic mass and CA-125 levels >65 IU/ml the test has a sensitivity of 97% and a specifity of 78%.

CA-125 and Surgery

Now a days carcinoma ovary is mainly treated with surgery and chemotherapy.Those patients with persistently elevated levels of CA-125 after three months of treatment most likely have resistant tumor. CA-125 levels should be interpreted with caution untill 3 to 4 wks after operation as often surgery itself results in peritoneal inflammation and may lead to rise of serum levels.Various studies have shown that postoperative serum CA-125 levels measured 3-6 wks after primary laporatomy were sifnificantly lower than the preoperative ones(6). Thus for invasive ovarian cancer and

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tumors of low malignant potential,preoperative and postoperative CA-125 levels may serve as important prognotic factors for assessing adequacy of surgery.

CA-125 and Chemotherapy

For ovarian cancer,pre chemotherapy CA-125 levels can accurately reflect tumor burden after cytotoxic therapy(4).Low serum concentration of CA-125 was found after the third course of chemotherapy in all patients with epithelial ovarian carcinoma without evident disease. In partients with progressive disease,positive CA-125 findings were found in 72% of the patients after the third and in 100% after the sixth course of chemotherapy. A response in CA-125 levels after the first two courses of chemotherapy may indicate which patients should continue with the current chemotherapy regimen and which patients should be offered salavage therapy. Patients with serum CA-125 values below the normal valueof 35 IU/ml after two courses had a significantly longer median survival and longer disease free survival, than did those patients whose CA-125 levels dropped to normal after the third or a later course of chemotherapy.

CA-125 and response to treatment

It is difficult to measure the response to chemotherapy without exploratory surgery in carcinoma ovary.It has been shown that definitions based on a 50% or 75% decrease of CA-125 levels have been shown reliably to define partial response of ovarian cancer, in patients receiving first line chemotherapy[7]. When initial tumour size, marker levels and time to achieve normal levels of CA-125 were combined complete and partial remission could be predicted in more than 85% of patients.

CA-125 and follow-up

The most important of CA-125 is in the assessment of patients for recurrent disease post-oophorcetomy[2]. Gard et al have proposed that a lower level of 15 iu/ml may be more useful as a cut off when following patients with a known history of ovarian carcinoma. The rationale for this is that patients in their study with a nadir level in the intermediate range (15-35 IU/ml) invariably developed recurrent disease[8].

High CA-125 concentration were noticed in 95.7% of patients with clincial signs of recurrences of the disease and it was proved at second-look surgery 7. Elevated CA-125 level preceded clinical diagnosis of relapse by 3 to 6 months but only in the patients who at earlier stages of the treatment also showed abnormal levels of the marker[9]. The monitoring of patients with complete remission who showed normal CA-125 level throughout the treatment was not fruitful[9].

A recommendation to increase the significance of raised serum CA-125 levels during follow-up is to define at doubling of CA-125 from the upper limit of normal as progression. Using this method sensitivity was 85.9%, specificity 91.3%, positive predictive value 94.8% and negative predictive value 77.8%.

CA-125 and Prognosis

In carcinoma ovary, CA-125 level and established predictors of disease have been compared using a cox multivariate analysis to determine their relative prognostic value. The CA-125 trend was found to be most significant variable followed by the FIGO stage. Serum level generally reflects volume of disease and high levels usually indicate unrecestability and a poorer prognosis[10]. Serial CA-125 measurements fairly accurately predict the patient survival.

Conclusion

A variety of tumour markers have been tried to assist in the successful management of epithelial ovarian cancers. At present serum CA-125 is the gold standard tumour marker for this disease. It helps in early detection, staging and assessment of adequacy of surgery and effectiveness of chemotherapy. It facilitates followup and predicts subclinical recurrence progression, prognosis and survival. Now-a-days, management of carcinoma ovary, cannot be envisaged, without CA-125 levels.

References

1. Kumar A. Significance of tumour markers in gynaecological cancers. Obs and Gynae Today 1999;4(2):82-85.
2. Aziz DC. tumour markers A clincial utility update. Laboratory 1998;1-5.
3. Toftager-Larsen K, Hording U, Dreisler A. CA-125 placental alualine phosphatase and tissue polypeptide atigen as preoperative serium markers in ovarian carcinoma. Gynae and obstet Today 1992;33(3):177-82.
4. Osta Poviez DM, Keit J, Perez CA. Principles and practice of radiation oncology Philadelphia Lippin Cott-Raven Publishers 1998;1853-1880. ·
5. Ozoh RF, Schwartz PF, Eifel PJ. Ovarian cancer Fallopian tube carcinoma and peritoneal carcinoma cancer Principles and practice of oncology Philadelphia. Lippincot-Raven Publishers 1997.1502-1540.
6. Makar AP, Kaern J, Kristensen GB, Vergote I. Evaluation of serum CA-125 level as a tumour marker inn borderline tumours of the ovary. Int. Journal of Gynaecol cancer 1993;3(5):299-303
7. Rvstin GJS, Nelstrop AE, Meelean P, Brady MF et al. Defining response of ovarian carcinoma to initial chemotherapy according to serum CA-125. J. Clin. Oncology 1996;14(5):1545-1551
8. Gard GB, Houghton CRS. An assessment of the value of serum CA-125 meansurements in the management of epithelial ovarian carcinoma. Gynaecol oncology 1999;53(3):283-289
9. Markowska J, Kubaszewska M. value of CA-125 as a marker of ovarian cancer. Eur. J Gynae Oncology 1992;13(4):360-365.
10. Young RC. Gynaecologic malignancies Harrison's principles of Internal Medicine. New York: McGraw Hill Publishers 1998;606-607

MINIMAL ACCESS SURGERY THE PORT SITE COMPLICATIONS.

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INTRODUCTION:

Around 200 years ago, endoscopy was first started with the help of a tin tube at the end of which a candle was placed to provide the illumination.[1] The marriage of technology with advances in surgical expertise was responsible for the third major revolution that took place in the 19th century the other two being Asepsis and Anaesthesia. The modern era of laparoscopy started in 1966 with the development of Hopkin's Rod System.

Ever since Phillipe Mouret performed the first laparoscopic cholecystectomy in 1987,[2] there has been a rapid explosion in this field. Initially met with resistance laparoscopic cholecystectomy has become the "Gold Standard" in the recent times for the treatment of cholilithiasis[3] As the list of surgeries undertaken by laparoscopic method started to increase by leaps and bounds, newer complications were encountered by surgeons. The experience of early workers was responsible for the framing of strategies for the management of these complications. In this article an effort has been made to apprise the young surgeons about potential complications met at the basic point of entry i.e port site.

PORT SITE COMPLICATIONS: -

These can be further grouped under two heading.
ACCESS RELATED COMPLICATIONS.
POST OPERATIVE COMPLICATIONS.

ACCESS RELATED COMPLICATIONS

The placement of Veress needle and the primary Trocar and cannula is done blindly, which results in maximum complication rate at this point. All the other ports are placed under vision, hence the overall incidence of complications is minimal at these sites.

Usually the creation of pneumoperitoneum is done by a Veress needle which is the basic design in majority of the needles. In majority of cases the design incorporates a spring loaded blunt tipped obturator which is advanced past a sharp needle tip as it enters the abdominal cavity.[4] The spring loaded system has the advantage of covering the exposed sharp edge of the needle as soon it penetrates the Abdominal wall. The reusable type of needle is most commonly used in our setup due to the lower cost. Over a period of time the needle tip becomes blunt requiring excessive force, which can result in injury.

It is advisable to check the needle each time before use to see whether the obturator covers the needle. Proper cleaning of the parts of the needle after the surgical procedure is over is also mandatory to prevent blood from coagulating & disrupting the spring-loaded mechanism. The needle also requires sharpening after every 50 punctures.

A significant advantage of the disposable needles is that the tip is always sharp standardizing the amount of pressure required for insertion.[4] Some designs where the manufactures have put a guard on the needle are also available.

The lifting up of abdominal wall, staying away from previous incision sites, insuffulation at the Palmer's point in the left hypochondrium and use of saline drop test all help in reducing the number of complications.

A special cuffed cannula (optical viewing trocar) in which the path of the entry can be visualized with the camera is also available which greatly reduces the incidence of complications[5]

The "Hasson's Open technique"[6] and the semi open technique using the dissected umbilical tube can also reduce the number of complications. Even though visceral injuries occur with the same frequency the incidence of catastrophic vascular injuries is vastly minimized.

During the creation of pneumoperitoneum the two main complications encountered are.

VISCERAL INJURY
VASCULAR INJURY.

VISCERAL INJURY

Visceral perforations occur in 0.1% to 0.3% of all laparoscopic procedures.[7] Veress injuries of the bowel are usually small and seal quickly, hence do not usually require any intervention. Usually the commonest visceral injury occurs to the small bowel.

If persistant leakage is noted on laparoscopy, sutures may be placed under guidance if the surgeon is well versed with intracorporeal technique of knotting otherwise the segment of intestine may be eviscerated through a small incision and the repair carried outside.

VASCULAR INJURY.

These can occur in two ways.

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EPIGASTRIC VESSEL INJURIES.
INTRA ABDOMINAL MAJOR VESSEL INJURIES.

EPIGASTRIC VESSEL INJURIES

These are the most common vascular injuries noted and usually occur due to blind insertion of a trocar cannula.

To prevent this it is advisable for the camera assistant to bring the tip of the laparoscopie near the area where the surgeon is expected to make the incision. The light from the tip delineates the vessels in the abdominal wall and they can be avoided..

If the vessel has been damaged, then temponade can be done by compressing the site with the trocar. Usually this is enough to stop the bleeding. If at the end of the procedure the area is still bleeding then direct suture ligation can be tried. In some cases a port closure needle may be required for closure of the bleeder.

INTRA ABDOMINAL MAJOR VESSEL INJURIES.

Injuries to major intraabdominal vessels may occur in 0.4% of all laparoscopic procedures.[7] Most of these injuries occur when the insufflation needle or when the first trocar is inserted percutaneously through the abdominal wall. To avoid this, the needle and the trocar should be aimed at an angle and caudal to the bifurcation of great vessels.

The "Hasson's open technique" vastly reduces the incidence of major intra abdominal vascular injuries.[7] Even the semi open technique using the umblical tube after dissecting it out minimizes the risk of intra abdominal vascular injuries.

Catastrophic complications include CO_2 embolism that is created by injection of CO_2 directly into the circulation as may happen when a vessel is cannulated. C.V.S. collapse quickly ensues, accompanied by cyanosis, increased venous pressure and ventricular arrhythmias. A decrease in end tidal PCO_2 occurs as the gas obstructs the right ventricular outflow tract. Prompt recognition, positioning the patient in head down and left lateral position, immediate cessation of the insuffalation are the most important steps to be taken. The Anaesthetist should remain with the patient all the time especially at the time of creation of pneumoperitoneum. Hyperventilation and aspiration of gas through C.V.P. catheter may also be required.

POST OP COMPLICATIONS:

These are classified under two headings.
MINOR COMPLICATIONS
MAJOR COMPLICATIONS

MINOR COMPLICATIONS

These usually do not require any active intervention and respond to conservative means like anti-septic dressings etc. There are usually no long-term sequelae. These include serous discharge, uneven wound margins, superficial necrosis at the skin edges and minor oozing of blood etc.

MAJOR COMPLICATIONS

1. Bleeding
2. Infections.
3. Hernias
4. Metastasis.

PORT SITE BLEEDING:

This may be very slow ooze, which can be seen on the overlying dressing post operatively. Pressure dressing is required on these areas.

Sometimes a bleeder in the margins and deeper sub cutaneous tissue may require Electro-coagulation. On some occasions the larger vessels (Superior/ Inferior Epigastric) may be torn during the insertion of Trocar & Cannula. As the bleeding is usually brisk it causes pooling and obscures the vision and also can track down the sides of Cannula to the site of surgical operation and this can cause confusion, hence some active treatment is required. It usually needs a ligature to be secured on to the bleeder or cauterizing it. A port closure needle popularly known as the shoemakers needle (Mochi's Needle) may be needed to go beyound the area to secure the bleeder.

In the post op period the bleeder can present as a port site Haematoma. The patient complains of more than expected pain at the site with swelling and bluish discolouration of the area. The area may need to be opened and the bleeder may be secured under a good light under local anaesthesia. Suction or a wick drain may be needed in the area.

PORT SITE INFECTIONS:

The trocar and cannulas and other instruments can either be autoclaved or chemically sterilized by Cidex (Glutarldehyde). It is imperative that the solution should be regularly changed and the minimum immersion time in the lotion is 20 minutes after proper washing and cleaning of the dismantled instruments under running water. Ethylene oxide gas plant sterilization is more effective method of sterilization.

It is also important to use medical grade CO_2 for pneumoperitoneum as industrial grade CO_2 is usually contaminated with bacteria and spores.

There are two types of port site infections.

Acute infections
Chronic infections

ACUTE INFECTIONS: This usually follows procedures in which the specimen is removed through the trocar sites. The contact of the potentially infected specimen leads to infection of the port site as in lap assisted appendicectomy.[8] To minimize this it is advisable to remove the specimen in an endobag. As the cost incurred by this is rather high, the inner covering of a drip set which is sterilized by E.T.O. plant is quite handy. Anecdotal reports using Condoms have also been described.

Usually the patients present with Sero-perulent discharge, pain and occasionally rise of temperature. Most common infections are staphylococcal in origin.

The management includes drainage of any underlying collection leaving the wound open. The patient is usually given second or third generation wide spectrum antibiotics.

CHRONIC INFECTIONS: In the third world countries where tuberculosis is prevalent, the patient can develop a chronic discharging sinus. Bhandarker O.S et al[9] reported about the port site infection with Mycobacterium Chelonei. Group IV atypical mycobacterium have been reported especially M. fortuitum and M. Chelonei with increased incidence. Investigations reveal non-caseating granulomas.

In these patients wide local excision of all sinuses and diseased tissues followed by secondary closure[10] is required. Patients also require Anti- Mycobacterial drugs.

The incidence can be decreased by proper sterilization of the instruments for 20 minutes in Cidex in between two cases.

PORT SITE HERNIAS:

Port site hernias are found with increasing incidence especially in cannula sites with diameter of 10 mm or more. It is rarely found in 5-mm cannula sites. This is one of the most dangerous complications even though it is quite rare in frequency. Some authors report the incidence to be around 1%.[11] Usually majority of the cases are of the Richter's type involving the small intestine. The type of hernia is responsible for the delay in diagnosis and the morbidity. Some researchers are of the opinion that the hernias usually follow a port site infection than otherwise.

To prevent this complication port site closure using the needle described above is essential. The results are quite excellent.[12] The needle takes the suture from the subcutaneous tissue to the one side into the peritoneal cavity under vision and then it is taken out from the other side to close the fascial defect.

If a port site hernia is encountered it can be managed by performing a relaparoscopy

To either close the defect or by placing a patch of non-absorbable mesh over the defect.

PORT SITE METASTASIS:

As the horizons of the laparoscopic surgery increased it was used in patients with malignant lesions. In the early 90's the published data indicated a very high incidence of port site metastasis. This was found in patients of Visceral and gynecological malignancies.[13] Initial reports suggested an incidence of around 20%.

However the current published data does not reflect this. Various reports indicate the metastasis to be in the range of 0.5%-1.3%. This is comparable to the standard open surgical procedures(0.8-1.6%).[14]

A review article regarding the port site metastasis postulated that metastasis is secondary to Local trauma, Tumour manipulation, biological properties of the tumour, Individual surgical skills and insufflation of the Gas. They reported 164 cases of port site metastasis in 90 published papers.[15] Another study reported an incidence of 1% in 1737 cases of colorectal cancer who underwent laparoscopic surgery[16]

At present it can be safely concluded that with the use of inert gas and proper surgical technique the port site metastasis is reduced to minimum and the overall survival rate is better in comparison to open surgery.[17]

REFERENCES

1. Knyrim K, Seidlitz H, Vakil H, et al. Perspectives in electronic endoscopy, past present and future of fibres and C.C.Ds in medical endoscopes. Endoscopy :1990 :22(Suppl) :2-8
2. Watters. R.C. Basic technique of laparoscopic cholecystectomy in Atlas of laparoscopic surgery eds: Pappas T.N, Schwartz. L.B., Eubanks. S,: Current medicine, Philadelphia, 1996: page 6.2
3. Soper N, Stockman P, Dunnegan D, et al. Laparoscopic cholecystectomy: The new "Gold Standard" Arch Surg: 1992: 127: 917-21.
4. D'Amico, Schwartz. L.B., Eubanks. S, Laparoscopic Instrumentation and Basic technique in Atlas of laparoscopic surgery eds: Pappas T.N, Schwartz. L.B., Eubanks. S,: Current medicine, Philadelphia, 1996: page 1.4
5. Katkhouda N, Advanced laparoscopic Surgery, Harcourt brace & Co. Asia; 1998 Chapter 1; page 10-11
6. Katkhouda N, Advanced laparoscopic Surgery, Harcourt brace & Co. Asia; 1998 Chapter 1; page 14.
7. Bonjer E.I, Hazebrock E.j, Kazemier G et al. Open and closed establishment of pneumoperitoneum in laparoscopic surgery. Br. J. Surg; 1997; 84; 599-602.
8. Ghoneimi K, Valla J.S, Limonne M et al. Laparoscopic appendectomy in children. Report of 1379 cases, Abstract A45; British Association of Paediatric Surgeons 1992 Annual Congress, Manchester.
9. Bhandarker D.S, Bhagwat S, Punjani R, Port site infection with Myc, Chelonei following laparoscopic appendicectomy. Indian. J. Gastroenterol: 2001: Nov 20(6) ; 247-8
10. Rappaport W, Dunington G, Norton L, et al, The surgical management of Atypical Mycobacterial soft tissue infections. Surgery; 1990; Jul; 108(1) 36-9
11. Holzinger F, Klaiber C,. Trocar site hernias, A rare but potentially dangerous complication of laparoscopic surgery. Chirug; 2002 Sep; 73(9) ; 899-904
12. Elashry O.M, et al. Comparative study of port closure technique following laparoscopic surgery. Jr Am Coll of Surg; 1996; 183(4) 335-44
13. Nauka C, Monson J.R.T, Darzi A,. Abdominal wall metastasis following laparoscopic surgery. Br. J. Surg; 1994; 81; 648-52
14. Fornar P. Port metastasis; fact or fiction. Urology; 2002; Mar; 41(2); 113-9
15. Scaff B, Paolucci V, Thomopoulos J. Port site recurrence after laparoscopic surgery, A review. Dig Surg; 1998; 15(2); 124-34
16. Zinora O, Gervaz P, Wexner S.D. Trocar site recurrence in laparoscopic surgery for colorectal cancers. Surg Endosc 2002; Apr ;16(4); 722-3.
17. Falk P.M, Beart R.W.Jr, Wexner S.D,et al. Laparoscopic Colectomy A critical appraisal. Dis. Colon. Rectum; 1993, 36, 28-34.

PRENATAL SEX DETERMINATION FEMALE FOOETICIDE AND INFANTICIDE

Tabasum Parvez

Prenatal sex determination can be done earliest by 10-11 weeks of Gestational age Here placental tissue sample is taken ultrasound guided known as chrionic villlus sampling and sent for chromosomal analysis to genetic laboratories. It carries 0.8% risk of miscarriage. It costs between Rs. 1000-1500 with 95-100% accuracy.

Amniocentesis started in 1974 for foetal birth defects, sex linked, genetic and chromosomal disorders. It was used widely for chromosomal analysis and sex determination at 15-16 weeks of Gestational age in genetic laboratories. Risk of miscarriage 0.5%

Ultrasound gives sex details at 16-18 weeks by experts. Most specific at 18-20 weeks of gestational age. Besides this, foetal anomallies and diseases are ruled out at this stage. On USG.

i) Trimester - streaks appear between the legs in case of female.

ii) Trimester - labia swells up because of hormones. Other methods are temperature dependant sex determiation (TSD) & DNA Analysis. Barr bodies in buccal musosa seen in females.
  - with sex chromatin positive obtained in 75% of cells.

Old Myths

i) Heart Rate - Heart Rate 140 or > 140/min it's a female Heart Rate - 139/min or less it's male.

ii) Chinese calendar test Here mothers date of Birth and month of conception was taken in consideration.

iii) Draino Crystals: To urine 3ml draino crystals were added (solution made) brown coloration was seen for females, green coloration was noted in males. Advances in Science was used for further female abuse. Abuse in terms of female foeticide and female infanticide is now as old as many cultures. Millions of gender selective deaths in history are seen and heard

Female foeticide began since 1978 when scientific methods determined tests for sex determination. Its origin was following amniocentesis used for various foetal well being tests. Female foeticide was seen more in higher classes and infanticide in lower class group of people

It's a critical problem in III world countries India, China, Pakistan and Bangladesh, especially in China & India. In India mostly in Tamilnadu and Rajasthan especially in villages and Tribal areas. Targets are female fetuses to avoid female children.

In greece the murder of female infants was so common that among 6000 families not mroe than 1% had 2 daughters.

In India young girls were murdered as a matter of course because of Hindu belief and rigid caste system. In some villages no girl babies were found.

In Tamil Nadu in 300 poor hamlet 196 girls died under suspicion. Some were fed dry unhurdled rice that punctured their wind pipes, swallowing poisonous powdered fertilizers, some strangulated or starved to death An incidence in Tamil Nadu where after the birth of IInd daughter the mother refused to nurse here baby for 3 days. The villages sympathizing with the mother and fedup with the cries of the baby, fed the baby with castor oil mixed with small quantity of milk. The baby bled from nose and other sources profusely and died soon after. A daughter always a liability and they thought how could the mother bring up another girl.

In Jaipur 3500 female foeticides per year on average seen and where killing is no sin.

UNICEF studies in Bombay conducted in 1984 showed 7999 out of 8000 Aborted foetuses were females..

In Rajasthan good nutritrious diet, health care and more money spent for clothing of boys than for girls or women

Tamil Nadu government finances parents who have 2 or more females and voluntarily undergo sterlization.

Statistics: Show that 85% high and Middle class women undergo sex determination because they can afford 50% are forced by husbands and their families 42 and of 50 gynaecologist in Bombay do sex determination 6 out of 42 do chorionic villus sampling. In India 19% of doctors see it as a wrong practice.

Sex determination is now a social problem like dowry and child marriage. No point in blaming doctors alone. This practice has been banned medically and morally. Its consequences will be altering male female ratio which can have serious impact in future

Campaign against sex determination & preselection was

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started first in 1985 in Bombay and other cities.

Private bills one in parliament and other in Maharashtra legislature.

a) Determining spread of sex determination tests and female foeticides in Bombay city.
b) Finding related aspects of this practice.
c) Doctors involved in such practice punishment for 5 years along with punishment for females. Govt. imposed ban on such tests w.e.f 1996 Jan i.e, no genetic tests can be done in any institution unless specifically recognized for this purpose by state govt

SRO-198. Prenatal determination of sex is an offence punishable under law Violation of law could lead to 5 years prisonment. In exercise of powers confered by subsection 3 of section I of J&K preconception and prenatal sex selection prohibition and regulation act 2002 It appointed 1st June 2002 as the date on which the said act came into force by order of J&K Govt. issued by department family welfare, Ministery of Health, Govt. of India.

References:

1. Banning Prenatal sex determination. The scope and the limits of Maharasthra legislation. A Reader Edited by Lakshmi Lingam 1998.
2. The Victimizing Discours: Sex determination Technologies and policies Arora, Dolly. Economic and political weekly Feb 17, 1996 page 420-424.
3. The Hindu: Prenatal tests. Edition of India's National News Paper Thursday April 11, 2002.
4. The Hindu warning of Prenatal diagnostic tests. India's National News Paper Wednesday March 06, 2002 (Business live. The sportstar. Frontiline)
5. Sex selection and Questions of law. Gender Issues, India's Natioonal Magazine The Hindu. Issue 21, Oct. 14-27 2000.
6. Sex determination tests: summons health secretaries of II Major states asked to personally present on January 29.

METASTATIC MALIGNANCY OF BONE (PART II)

A.R.Khan M.D.

Abstract: *FNAC over 11 ½ year period (Jan 1991-June 2002) revealed 110 bone tumors, including 29 metastatic cancers. Lymphomas were excluded. Added to this is one case of metastatic paraganglioma to sternum that was recorded subsequently. There were 22 cases of Adeno-Carcinoma, 3 cases of Small Cell Anaplastic Carcinoma, 3 cases of Follicular Carcinoma Thyroid, 1 case of Undifferentiated Large Cell Carcinoma and 1 case of Paraganglioma. Histopathology comparison was available in all but one case of Large Cell Undifferentiated Carcinomao and one case of Paraganglioma. All but one case of Adeno-Carcinome, were correctly diagonosed on FNAC. This case of metastaticAdeno-Carcinoma upper cervical spine was misinterpreted as Chordoma. In 12 cases of Adeno-Carcinomas including 5 with known cancer, primary was suggested/ consistent with the known primary tumor. Smears in 3 cases of Small Cell Anaplastic Carcinoma, 3 cases of Follicular Carcinoma Thyroid, 1 case of Undifferentiated Large Cell Carcinoma and 1 case of Paraganglioma were digonostic to suggest the primary. Overall sensitivity of FNAC digonosis of skeletal metastsis were 96.4%.. Diagnosis of skeletal metastatic cancer by FNAC is simple non-invasive procedure in patients with known and unknown cancers to determine the staging and institution of appropriate therapy.*

Key – words : *FNAC Bone tumor, Metastatic Bone cancer*

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INTRODUCTION:

Metastasis is the most frequent bone cancer that occurs in about 30% of patients with cancer[1,2]. Any site may be involved but the most frequents sites are the axial skeleton, proximal femur and proximal humerus[3]. In adults carcinomas particularly adeno-Carcinomas are most frequent, whereas Sarcomas are rare. Most of the skeletal metastasis originate from breast in females and prostate in men followed by Carcinoma lung and kidney in either sex[4]. In children the most frequent cancers metastasing to bone are Neuroblastoma, Wilm's tumor and Rhabdomyosarcom. Lytic bone lesions for suspected metastasis have been the main targets of FNAC since early 1930s [5].

Author reports here his experience on FNAC of metastatic bone disease over a period of eleven and a half year (Jan 91- June 2002)

MATERIAL AND METHODS:

These have been discussed in part I of the series.

The cytologic criteria for diagnosis of metastatic bone cancers are available in various texts on Cytology [6,7].

RESULTS:

FNAC over 11 ½ years revealed 110 bone tumors, including 29 metastatic bone cancer (Table I & II). Excluded from the analysis are 4 cases of disseminated Non-Hodgkin's Lymphoma , diagnosed initially by FNAC of bone (Table III).Added to this series is an interesting case of Paraganglioma metastatic to sternum diagnosed on FNAC (Case 30, Table II).

As shown in the table II, the age of the patients varied between 31-85 years with 22 males and 8 females, 21 were located in the axial skeleton and 9 in the long bones. All the patients had history of local pain/swelling and osteolytic/ osteoblastic lesions on x-rays. Table IV summarizes the cyto-histopathology correlations.Cytology of the smears is shown in Figs 1-8A. Thus it can be seen that there were 22 cases of Adeno-Carcinoma, 3 cases of Small Cell Anaplastic carcinoma, 3 cases of Follicular Carcinoma Thyroid, 1 case of Undifferentiated Large Cell Carcinoma and 1 case of Paraganglioma. Out of 22 cases of Adeno-carcinoma, diagnosis of Adeno-carcinoma was correctly made in 21 cases with 1 incorrect diagnosis (case.10) of Chordoma for mucinsecreting Adeno-carcinoma in upper cervical spine.

In 5 cases (cases 6,14,16,25 and 27) primary could be suggested. In another 5 cases Cases 08,12,17,20,21) smears were compatible with metastasis from known primary sites. In another 2 cases (case 3 & 19) imaging showed large masses in the lung and pancreas respectively, to suggest a possible primary. In 10 cases (cases 1,2,5,10,11,18,22,24,28,29) there was no clue to primary

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The First Part of this special series article, utility of fine needle aspiration cytology in the diagnosis of primary malignant bone tumors, appeared in JK-Practitioner 2003;10(2):143-151

Metastatic Malignancy of Bone

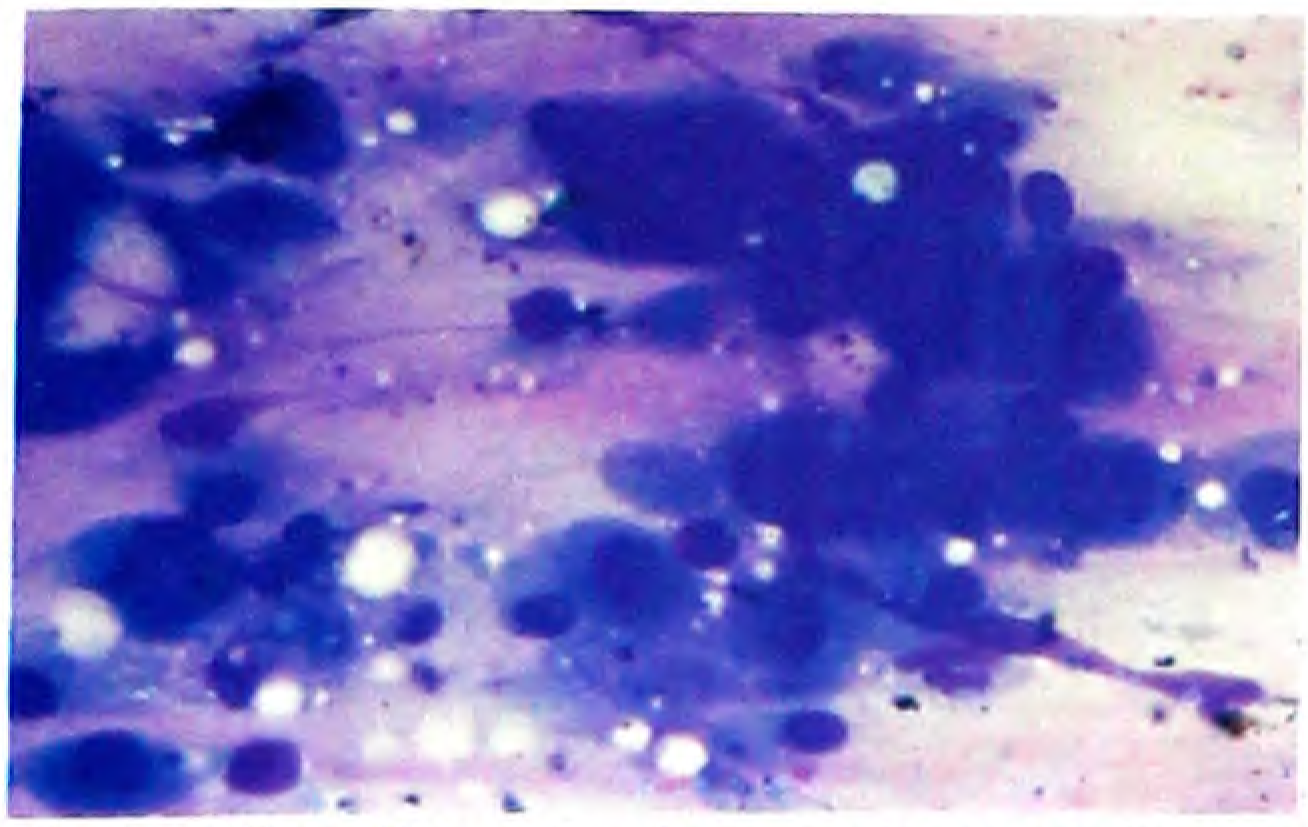

Fig 01. FNAC; Metastatic adeno- carcinoma mucinous to upper cervical spine. MGG x 400.

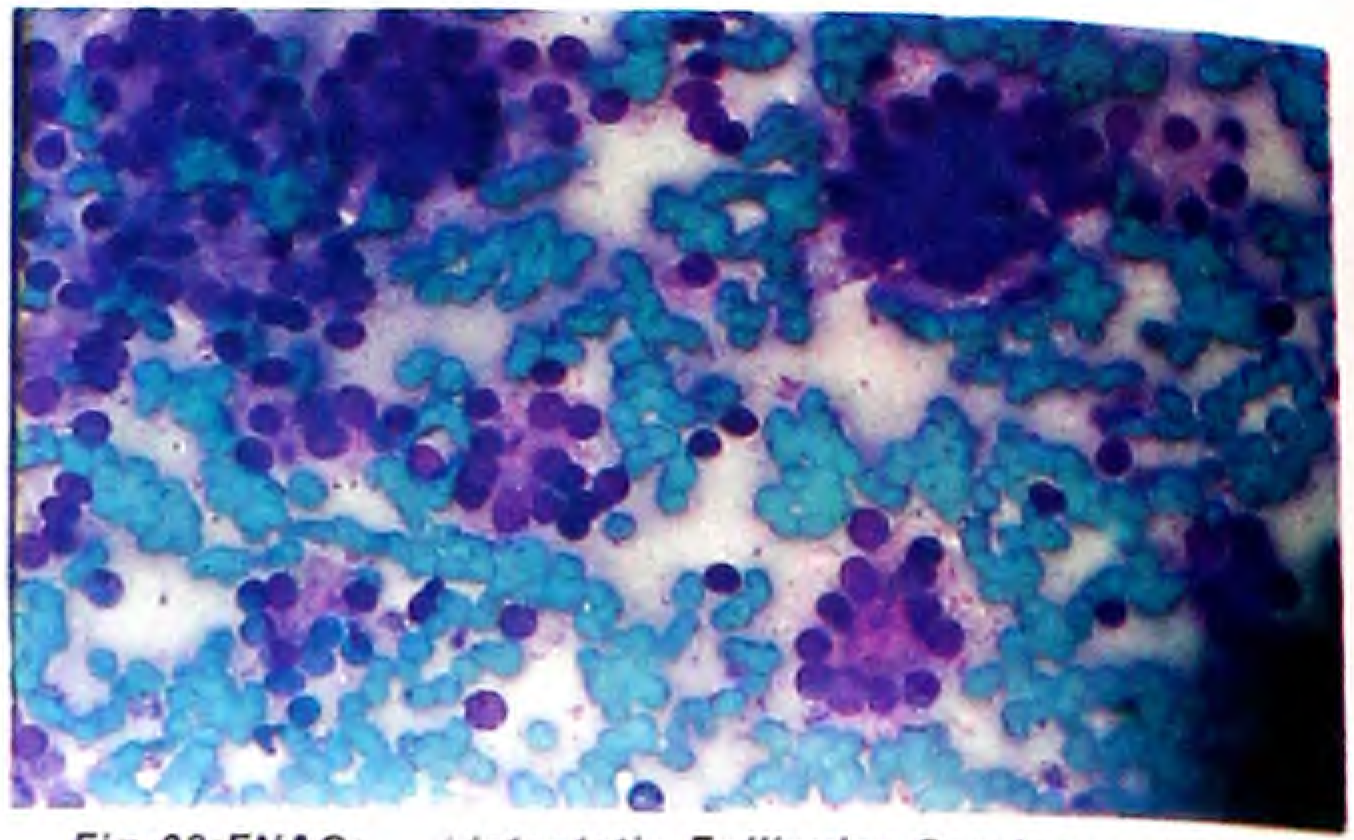

Fig 02:FNAC: Metastatic Follicular Carcinoma Thyroid.. Smers from pubic ramus shows well formed follicles, & the colloid in the center. MGGx 400.

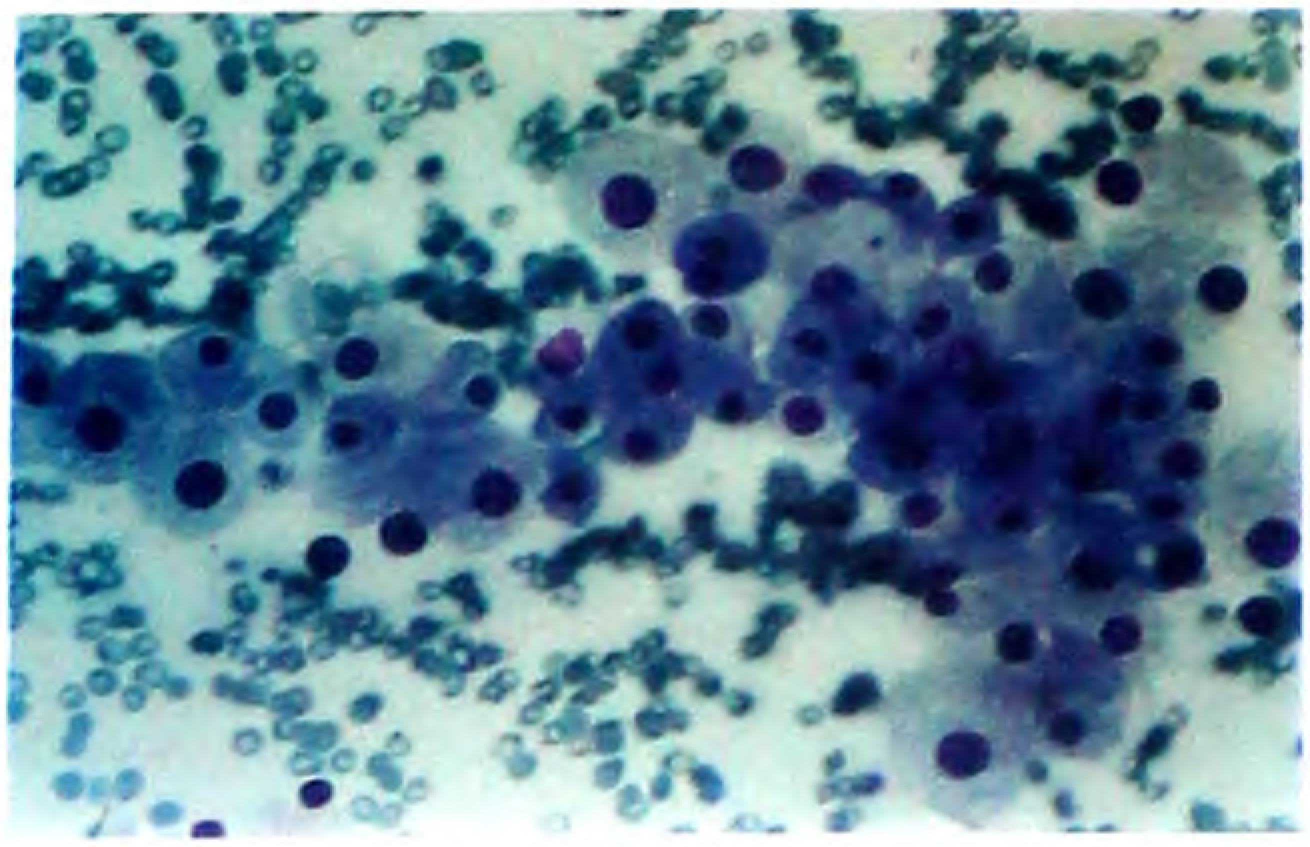

Fig 03:FNAC: Metastasis to humerus from Adeno-Carcinoma Kidney. Tumor cells in flat sheets show round nuclei with little variation in size, & abundant pale vacualated cytoplasm MGG* 400.

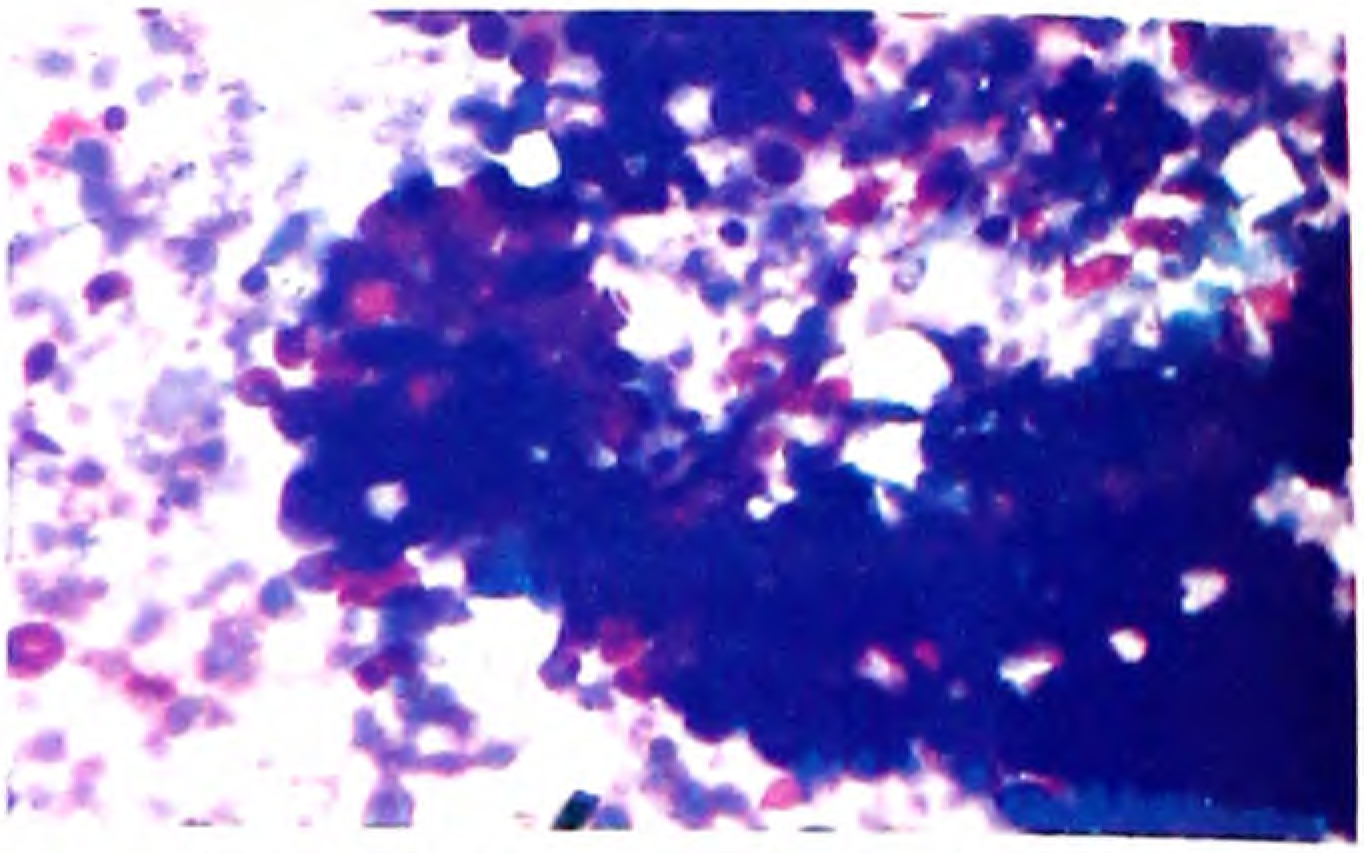

Fig 04. FNAC Smears from metastatic Adeno –Carcinoma Prostate to ileum, shows tight clusters of small cells, with little variation in tumor cells MGG x 400

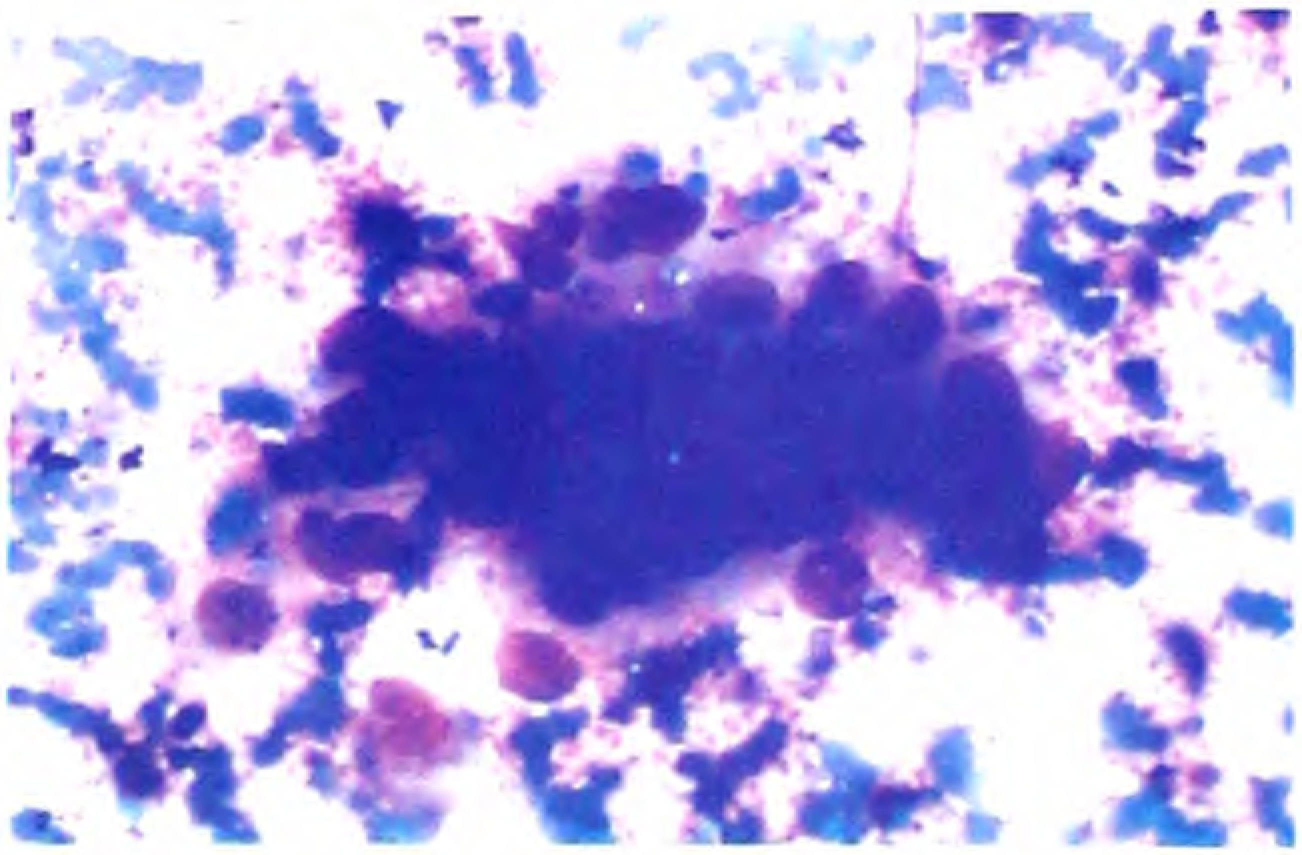

Fig 05. FNAC Metastasis to bone from Carcinoma Stomach shows clusters of tumor cells with eccertric hyperchromatic nuclei & purpish mucinous material in cytoplasm (signet ring cells) MGG x 400.

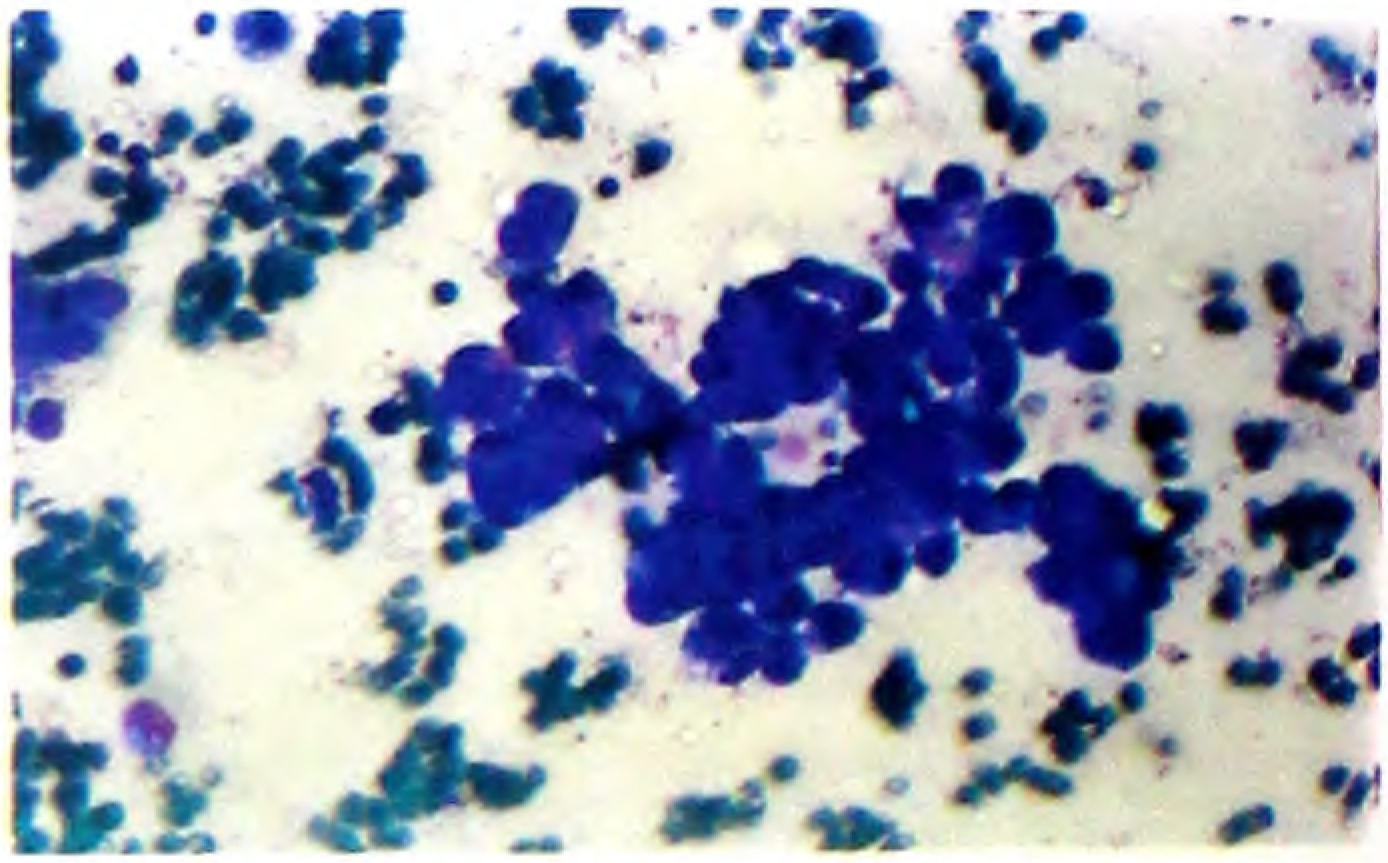

Fig 06: FNAC: Metastasis to iliac bone, with a cluster of Large Undifferentiated Carcinoma cells. Rt lung showed a large mass MGG x 400.

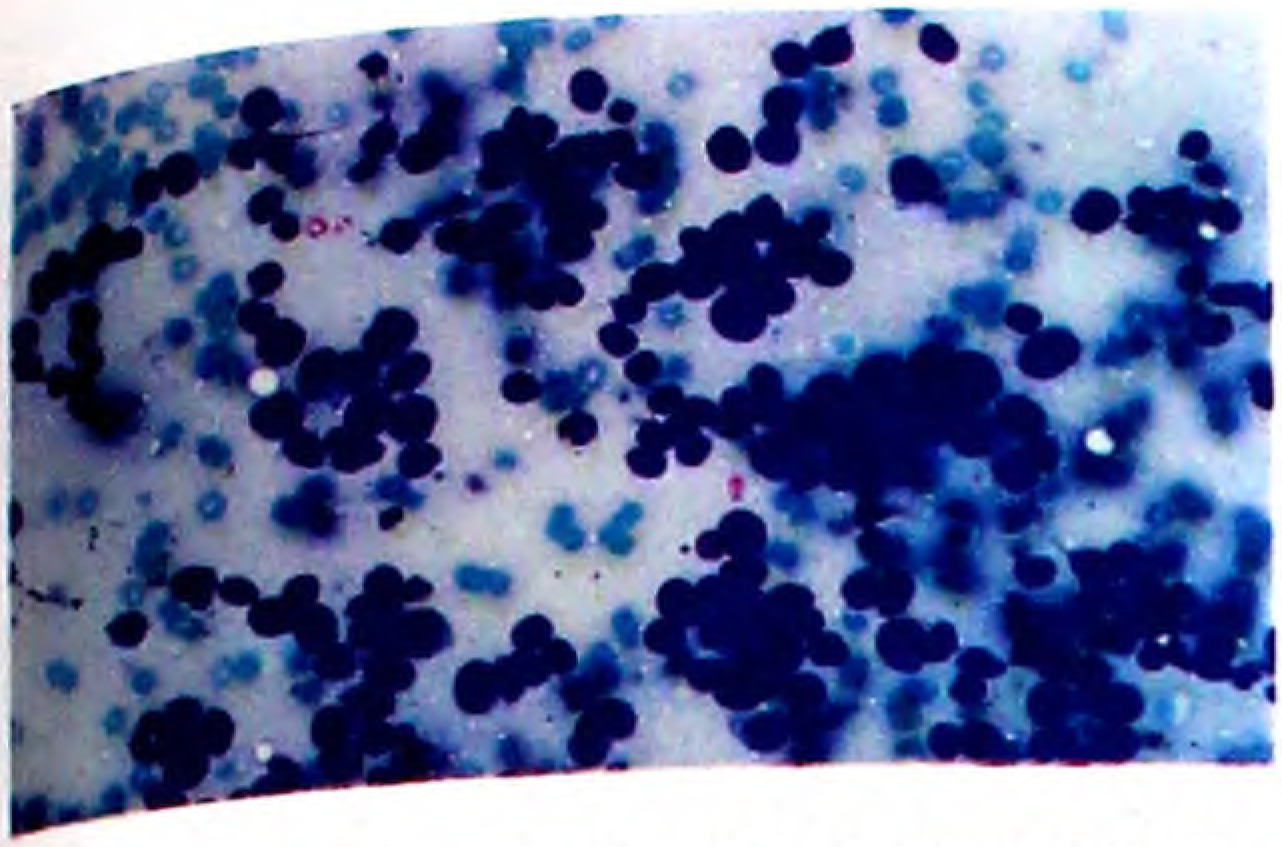

Fig 07:FNAC: Metastatic Small Cell Anaplastic Carcinoma rib shows small tumor cells with hyperchromatic nuclei scanty cytoplasm, arranged in clusters,poorly formed acis & Indian-file arrangement MGG x 400.

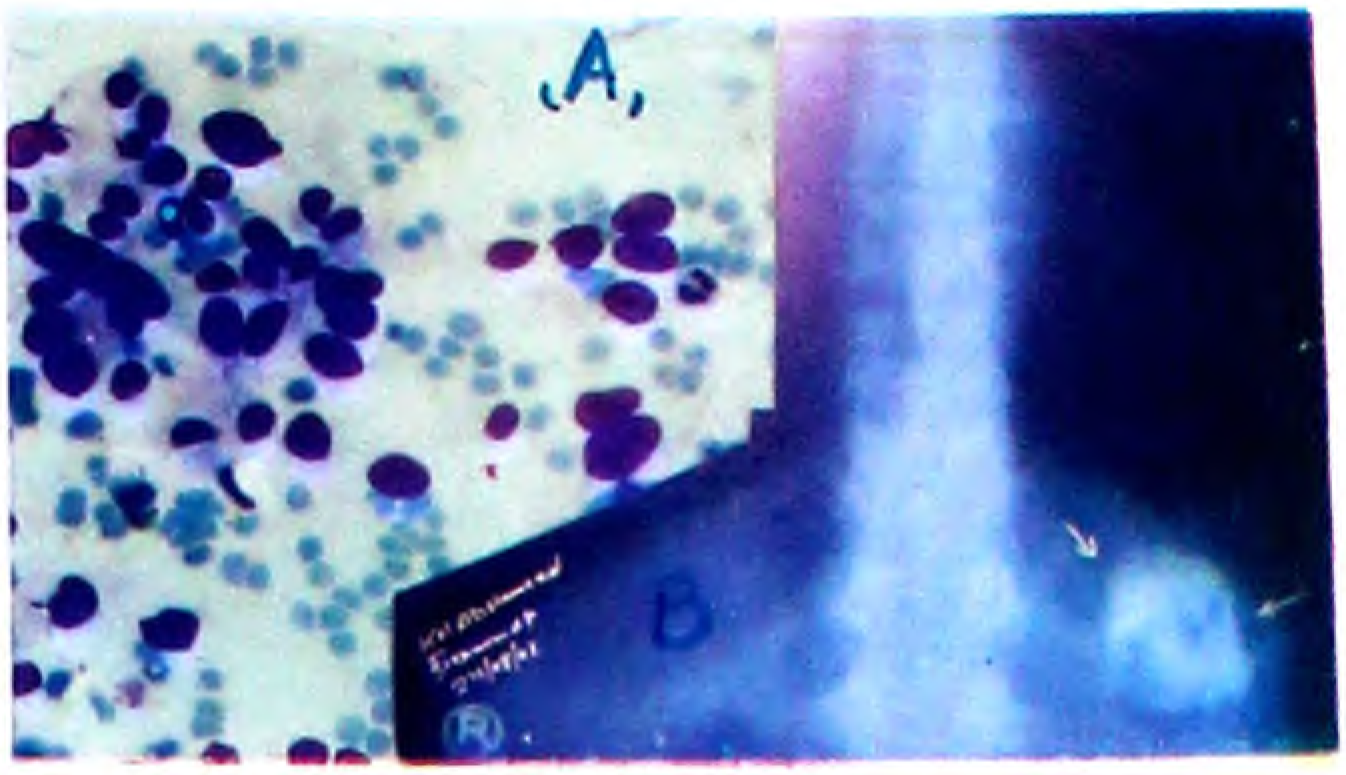

Fig. 08(A) FNAC: Metastatic Paraganglioma to sternum. Round to elongated cells with almost naked nuclei showing considerable variation arranged in small graps, dispersed & acinar arrangement on right hand side and clean background MGG x 400.

08 (B)Plain X-Ray –abdomen of same patient (8A) shows tumor in the left adrenal.

patients had history of local pain/swelling and osteolytic/ osteoblastic lesions on x-rays. Table IV summarizes the cyto-histopathology correlations.Cytology of the smears is shown in Figs 1-8A. Thus it can be seen that there were 22 cases of Adeno-Carcinoma, 3 cases of Small Cell Anaplastic carcinoma, 3 cases of Follicular Carcinoma Thyroid, 1 case of Undifferentiated Large Cell Carcinoma and 1 case of Paraganglioma. Out of 22 cases of Adeno-carcinoma, diagnosis of Adeno-carcinoma was correctly made in 21 cases with 1 incorrect diagnosis (case.10) of Chordoma for mucinsecreting Adeno-carcinoma in upper cervical spine.

In 5 cases (cases 6,14,16,25 and 27) primary could be suggested. In another 5 cases Cases 08,12,17,20,21) smears were compatible with metastasis from known primary sites. In another 2 cases (case 3 & 19) imaging showed large masses in the lung and pancreas respectively, to suggest a possible primary. In 10 cases (cases 1,2,5,10,11,18,22,24,28,29) there was no clue to primary on FNAC. Smears in 3 cases of Small Cell Anaplastic carcinoma and 3 cases of metastatic Follicular Carcinoma of thyroid were fairly characteristic to suggest the primary. After FNAC diagnosis of paraganglioma, imaging showed a tumor in the left adrenal(Fig 8B). Histopathology comparison from the metastasis/primary was available in all but the two cases including Large Cell Undifferentiated Carcinoma and Paraganglioma.

Table IV: FNAC- Metastatic Malignancy, Bone

	Cytology		Histopathology	
01.	Adenocarcinoma	21 cases.	Adenocarcinoma	21 cases
02.	Chordoma	1 case	Adeno-carcinoma	1 case
03.	Small cell Anaplastic Carcinoma	3cases	Small Cell Anaplastic Carcinoma	3 cases 3cases
04.	Follicular carcinoma Thyroid	3 cases	Follicular Crcinoma Thyroid	3 cases
05.	Undifferentiated Carcinoma Large Cell	1case	—-	
06.	Paraganglioma	1 case	—-	

DISCUSSION:

FNAC appears most useful in the diagnosis of bone metastasis in addition to some low grade and high grade primary bone tumors [8]. The main aim of FNAC is to establish the diagnosis of suspected metastasis and an attempt to characterize the primary to determine the stage of the disease and appropriate therapy. Metastasis to bone may be seen in patients of known cancer or be the manifestation of an occult primary .

Skeletal metastasis are generally multifocal. However, Carcinomas of the Kidneys and Thyroid are known to produce solitary lesions . Melanomas and Adrenal Neuroblastomas sometimes give rise to extensive skeletal

Table II- FNAC-Metasttic Malignancy - Bone

S. No	Age/Sex	Anat site	FNAC	Histopath	Primary
01.	49/M	Humerus-UE	Adeno-CA	Adeno-CA	—
02.	55/F	Humerus-UE	Adeno-CA	Adeno-CA	—
03.	60/M	Iliac-crest	Large Cell Undifferentiated-CA	—	? Lung
04.	60/F	Pubic ramus	Follicular CA Thyroid	Follicular CA Thyroid	Thyroid
05.	40/M	Mandible	Adeno-CA	Adeno-CA	—
06.	65/M	Humerus-UE	Adeno-CA Clear Cell	Adeno-CA Clear Cell	Kidney
07.	85/M	Ribs	SCAC	SCAC	Lung
08.	45/F	Ribs	Adeno-CA	Adeno-CA	Breast
09	55/M	Ribs	SCAC	SCAC	Lung
10.	60/M	Cervical Spine	Chordoma	Mucinous Adeno-CA	—
11	75/M	Clavicle	Adeno-CA	Adeno-CA	—
12.	58/M	Ribs	Adeno-CA	Adeno-CA	Adeno-CA II Part Duodenum
13	70/M	Scapula	Adeno-CA	Adeno-CA	? Lung
14.	75/M	Sternum	Adeno-CA Prostate	Adeno-CA Prostate	Prostate.
15.	65/M	Iliac crest	Follicular-CA Thyroid	Follicular –C Thyroid	Thyroid
16.	65/M	Lumberspine	Adeno-CA Prostate	Adeno-CA Prostate	Prostate
17.	60/M	Humerus-LE	Adeno-CA (Signet Ring)	Adeno-CA	Stomach
18.	55/F	Femur-LE	Adeno-CA	Adeno-CA	—
19	78/F	Humerus LE	Adeno-CA	Adeno-CA	? Pancreas
20.	31/F	Pubic ramus	Adeno –CA mucinous	Adneno -CA mucinous	Colon
21	60/M	Humerous-UE	Adeno-CA	—	G/E junction
22.	50/M	Scapula	Adeno-CA	Adeno-CA	—
23 .	54/F	Scalp	Follicular-CA thyroid	—	Thyroid
24.	60/M	Scalp	Adeno-CA	Adeno-CA	—
25.	60/M	Ischium	Adeno-CA prostate	Adeno-CA	Prostate
26.	45/M	Sclap	SCAC	SCAC	Lung
27.	75/M	Iliac bone	Adeno –CA prostate	Adeno -CA prostate	Prostate
28.	49/M	Femur UE	Adeno -CA	Adeno –CA	—
29.	42/M	Femur UE	Adeno -CA	Adeno-CA	—
30.	70/M	Sternum	Paraganglioma	—	—

involvement. Ewings sacrcoma and Osteo-sarcoma have a certain propensity to metastasize to other regions of the skeletal system. The bones most frequently involved are the spine, pelvis, femur, skull, ribs and humerus [4].

FNAC diagnosis of metastatic bone cancer has been facilitated with the presently available imaging techniques which can target the deeply situated and small bone lesions. The larger and superficial lesions can however be aspirated with the help of radiographs alone. The metastatic bone lesions have been the main target of FNAC, much earlier than the FNAC of primary bone tumor that took off in mid 1960's. The diagnosis of metastatic bone disease on FNAC is much more easier than primary bone tumors. A number of publications are available on the subject [8-20]. There are also some case reports of unusual tumors metastasing to bone [21-25] or metastasis to unusual sites [26-27]. An accuracy of 78.4% to 97% is reported in various seriese [12-14,17].

Table III: *FNAC-Metastatic Malignancy, Bone Non-Hodgkin's Lymphoma*

S.No	Age/Sex	Anat Site	FNAC	Histopath
01	40/F	Rib & Soft tissue	LCL	LCL
02	65/M	Ischium	LCL	LCL
03	70/M	Iliacbone	LCL	LCL
04	45/M	Ribs & Soft tissue	LCL	LCL

In the present series a total of 30 cases of metastatic cancers to bone diagnosed on FNAC are reported. The study excludes the 4 cases of Non-Hodgkin's Lymphomas, though disseminated, but diagnosed initially by FNAC (Table cases of metastatic Adeno-Carcinomas, 21 III).Out of 22 were correctly diagnosed with one incorrect diagnosis (Case 10,Fig 1) of Chordoma for mucinous Adeno-carcinoma. The cytology of these tumors, as Adeno-Carcinomas, was not difficult to interpret.Smears of metastatic Small Cell Anaplastic C Carcinomas (Fig 7) were fairly characteristic to suggest the primary. Though known to arise from a variety of sites, lungs remain the most common primary site of Small Cell Anaplastic Carcinoma.Smears of the metastatic Follicular Carcinomas Thyroid (Fig 2) were fairly diagnostic to interpret and suggest the primary.Smears of the metastatic Paraganglioma may not be easy to interpret unless one is well versed with the cytology of these rare tumors. The author with wide experience with FNAC of these tumors[28] had no difficulty to suggest the diagnosis. Imaging of abdomen subsequently revealed a tumor in left adrenal (Fig 8 B)

In summary FNAC diagnosis of lytic/sclerotic bone lesions of metastatic cancer is very useful to establish diagnosis in a patient with known or unknown malignancy with the aid of modern imaging facilities. This is an easy procedure compared to an open biopsy in cancer patients to determine the stage and appropriate therapy.

References

1. Johnston AD : Pathology of metastatic tumours in bone. Clin Ortho Related Res 1970:73:8-32
2. Healy J.H. Turnbull ADM Miedema B Lane JM Acrometasistasis J Bones Joint Surg.1986:68-A:743-46
3. Rosenberg A. Bones, Joints and Soft tissue Tumours. In Robbins Pathologis basis of disease ED. Cotran, Kumar Collins. Saundurs 6th edition 1999,pp1245-46
4. Akerman M, Waleas L. Bone in Fine Needle Aspiration Cytopathology.Ed. Young, GA, Blackwell Scientific Publications 1993. pp. 267 – 281.
5. Stewart FW.The dignosisof tumours by aspirations biopsy. Am J.Pathol 1933;9.801-12.
6. Orell SR Sterrett GF Walters Max N-1. Whitaker D.Edit Mannal & atlas of fine Needle Aspirations Cytology. 11 Edit Churchill Livingstine.
7. Young J A Edit. Fine Needle Aspiratin Cytopathology Blackwell Scienticatin Publication 1993.
8. Layfield LJ Armstrong K Zaleski S Eckardt J. Diagnostic Accuracy & Clinical Unility of Fine- Needle Aspiration Cytology in the Diagnosis of Clinically Primary Bone Lesions. Diagn Cytopathology 1992 9 (2),168 – 173.
9. Coley Bl, Sharp GS, Ellis EB. Diagnosis of bone tumours by aspiration.Ann J. Surg. 1931: 215 – 224.
10. Synder RE Coley BI. Further studies on the diagnosis of bone tumours by apiration biopsy. Surg Gynecol. Obstet 1945:80;517 – 522.
11. Ottolenghi CE: Diagnosis of orthopaedic lesions by aspiration biopy Result of 10,061 punctures.J Bone join Surg. 1955, 37 – A :443 – 464.
12. Stromby N Akerman M: Cytodiagnosis of bone lesions by means of Fine Needle Aspiration Biopsy. 1973 17 : 166 – 173.
13. Adler o Rosnberger A :Fine needle aspiration biopsy of osteolytic metastatic lesions Am J Radio 1979 133: 15 – 18.
14. El Khoury G Y Terepka RH Mickeleson MR. Fine needle aspiration of bone joint Surg. 1983. 65A: 522 – 525.
15. Agarwal PK Wahal KM Cytopathologic study of primary tumours of bone & joints.acta Cytol 1983: 27: 23 – 27.
16. Siddiqui S. Fanning CV. Fine needle aspiration of bone a one year experience. Acta Cytol 1988 32: 744 – 745.
17. Bennett JD Yacyshyn BJ Haddad RC Lefcoe MS Fine needle aspiration of bone lesions. J Canadian Assoc Radiology 47: 65 – 68, 1990.
18. Kabukcuogsu F kabukcuoglu Y kuzgun U-evren I. Fine Neede Aspiration of Malignant Bone Bennett lesions.Acta cytol 1998 42: 875 – 882.
19. Wedin R Bauer HC, Lambert S Soderlund V. Tani E. Cytologic diagnosis of Skeltal lesions. Fine needle aspiration in 110 tumour. J Bone Joints urg 2000 828: 673 – 678.
20. Jambhekar NA Moonim MT, Khatib Y. Bone FNAC-Expectations & limitations.A Review part 11 J of Cytol 2001 . 18 (1) : 1 – 7.
21. Ng IOL Leung C Y HO JWC: Fine needle aspiration cytology diagnosis of a symptomatic bony metastasis from an occult hepatocellular carcinoma.A case report. Diag Cytopathol 1990 6: : 127 – 129.
22. Pitman MB Thor AD Goodman AL Rosenberg AE. Benign metastasizing pleomorphic adenoma of the salivary gland diagnosis of bone lesions by Fine Needle Aspirations Cytology. Diagn Cytopathol 1992 8:384 – 387.
23. Thirumala SD Putti TC Medali NS. Wasserman PG: Skeletal metastasis from a granulosa cell tumur of the ovary.Report of a case diagnosed by fine needle aspiration cytology. Diag Cytopathol ,1998 19: 375 – 377.
24. Vadmal MS Smilari TF Hajdu SI. Fine needle aspiration features of a metastatic heptocellular carcinoma representing as a symptomatic bony lesion. Acta Cytol 1998 42 :1046 – 1048.
25. Huges JH Jenson Cs Donnelly Cohen MB. Silverman JF GeisingerKR Raabss.In role of fine needle aspiration cytology in the evaluation of metastaticclear cell tumours. Caner Cytpahol 1999 87 :380 – 389.
26. Vaezy A Budson DC : Phalangeal metastasis from branchogenic carcinoma.JAMA 1978: 239, 226 – 227.
27. Knapp D: Abdul-Karim F.W. Fine needle aspirations cytology of acrosmetastasis A Report of Two cases . Acta Cytol 1994 38: 589 – 591.
28. Khan A.R.. Fine Needle Aspiration Cytology of Head & Neck Paragangliomas .J of Cytology 2003: 20 (1) : 28 – 30.

FNAC- GIANT CELL TUMOR OF BONE A STUDY OF 13 CASES (PART III)

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ABSTRACT: *Analysis of FNAC of bone tumors over over a period of 11 ½ years (Jan 1991 – June 2002) revealed 13 cases of Giant cell tumors. Histopathology was available for comparison in all the cases. On FNAC 12 cases were diagnosed as Giant cell tumors and one case as Aneurysmal bone cyst. The histopathology showed 11cases of convevtional giant cell tumors, one case of Giant cell tumor with secondary Aneurysmal bone cyst and one case as primary malignant Giant cell tumor (G C T and fibrosarcoma). Thus the overall sensitivity was 92.4% and Specificity of 88.4%. 11cases were originating from long bone with typical epiphyseal location in 10 cases and metaphyseal in one case. Remaining 2 cases originated from metacarpals. FNAC smears in the context of clinical data and radiographic finding were characteristic of Giant cell tumor, comprising of closely intermixed, 2 cell population of mononuclear spindle and giant cells of osteoclast type. Other giant cell lesions, tumor and tumor like which need exclusion before making diagnosed of Giant cell tumor, have been discussed.*

The study proves that the pre- operative value of FNAC diagnosed of Giantl cell tumor bone in the context of clinical data and radiographs can avoid more invasive procedure of open or needle core biopsy.

Key- Words : *FNAC Primary bone tumors, Giant cell tumor (GCT)*

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INTRODUCTION

Giant Cell tumors (GCT) of bone is a tumor of unknown histogenesis constituting about 5% to 8% of all bone tumors[1-3]. Though seen over a wide age range of 10-70 years, most cases occur in the third and fourth decades with slight female predominance [1-3]. The most frequent sites are ends of the long bones[1-3]

Diagnosis of GCT bone is generally made on open and needle biopsy. However pre-operative FNAC diagnosis is now well documented in several publications [4-13] with obvious clear advantages over both open and needle core biopsies.

Material & Methods

A retrospective analysis over a $11^{1/2}$ years (jan 91- june 2002) reveated 110 bone tumours including 13 cases of giant cell bone tumours. The aspirations were conducted with the help of radiograps. A 20 ml disposable syringe with & without syringe holder 23 – 18 gauge disposable needles. The material aspirated was smeared on 5 – 8 slides. M.G.G &Haematoxylin stains were used regulary on all slides. Some slides were reserved for any special studies.

Results

The clinical data, FNAC and histo-pathology of 13 cases are shown in table 1. Age of the patient varied between 18 & 50 years though most cases (9/13) were seen in third and fourth decades. There were seven males and six females. 11 cases were from the ends of long bones (10 from epiphyseal region & 1 from metaphysis) and 2 from the metacarpals. 12 cases were diagnosed as GCT & one case as aneurysmal bone cyst. Histiopathology showed typical conventional GCT in 11 cases, primary malignant GCT (conventional GCT & fibro-sarcoma) in one and GCT with secondary aneurysmal bone cyst, in one case..

Cytologic findings:

The smears were haemorrhagic and all but one case revealed adequate cellular material. There were two cell populations, mono-nuclear and giant cells mostly closely inter-mixed. The monomorphic mononuclear cells disposed in small and large clusters and dispersed were spindle to ovoid with moderate amphophilic granular vacuolated ill-defined cytoplasm, ovoid nuclei with bland chromatin and 1-2 indistinct nucleoli. The giant cells mostly inter-mixed with mononuclear cells but also dispersed showed abundant dense basophilic fuzzy cytoplamic margins and multiple nuclei (>15/cell) with morphology of nuclei of mononuclear cells. The background was clean. (Figs. 1 & 2).

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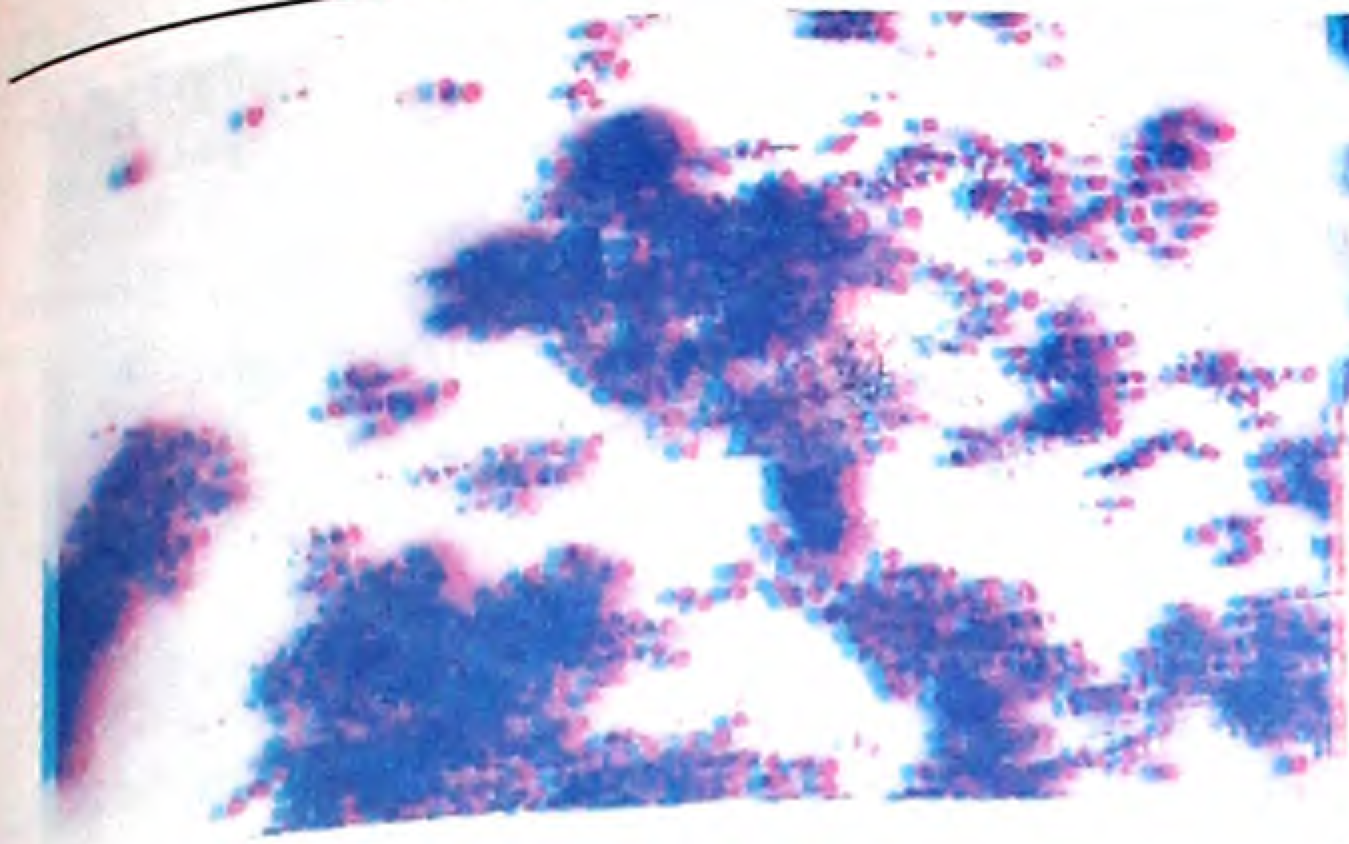

Fig 01. FNAC- Giant cell tumour bone – shows closely intermixed mononuclear & giant cells of oste clast type MGG x 200

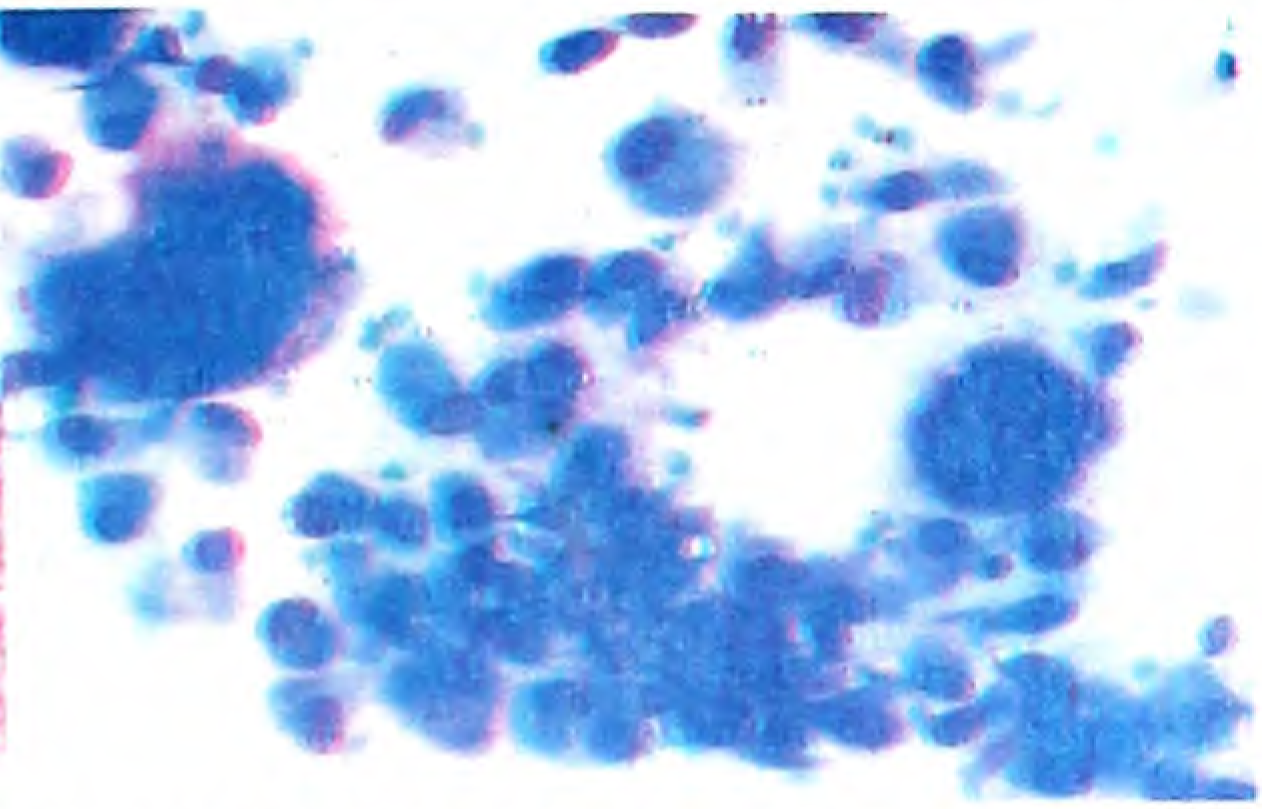

Fig 02. FNAC- Higher power view of fig 01 to high light details of monocular cells &giants cells.Polyhedral to spindle mononuclear tumour cells are uniform with a benign looking ovoid nuclei & moderate light basophilic cytoplasm. Giant cells show numerous overlapping nuclei & amphophilic ragged cytoplasmic margins. MGG x 400.

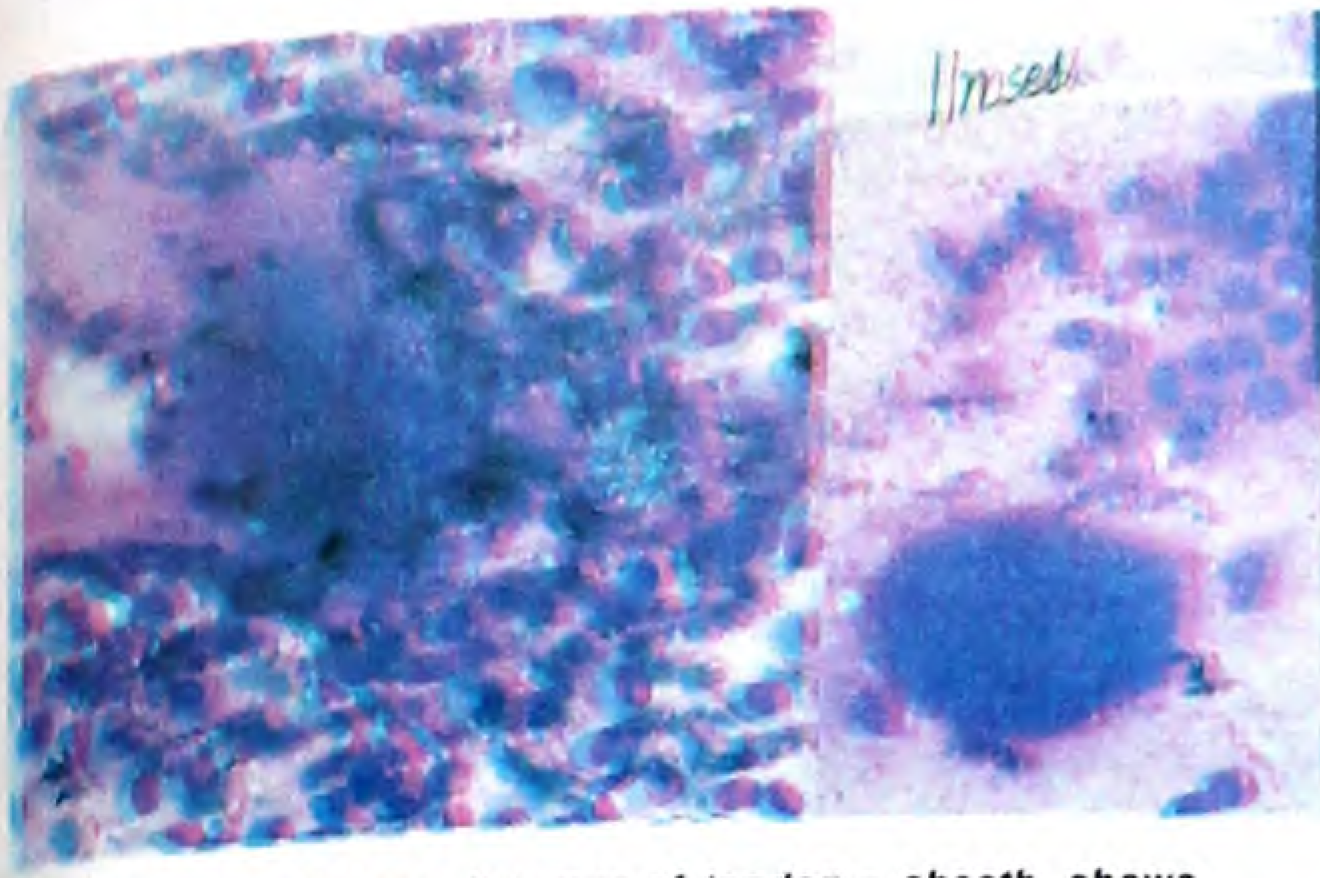

Fig 03. FNAC- Giant cell tumor of tendon – sheath, shows mono-nuclear cells with benign nuclei and scanty, ill defined cytoplasm.Eosiophilic homogenous material with superimposed nuclei (collagen).Inset show mononuclear cells and giant cell.Giant cell is morphologically indistinguishable from Osteoclast of GCT MGGx400.

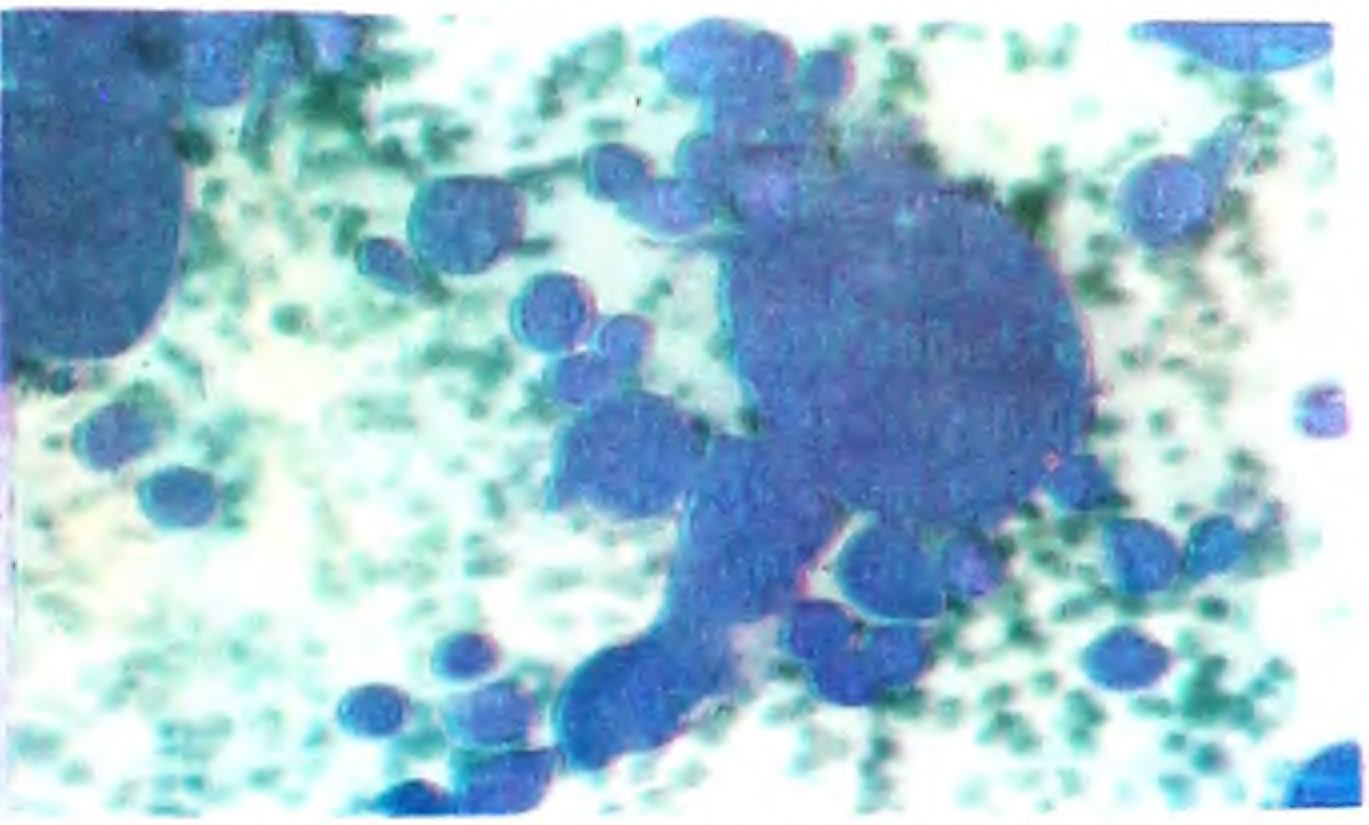

Fig 04. FNAC- Giant cell rich Osteo-sarcoma.The mononuclear tumor cells show striking malignant features in total contrast with mononuclear cells of GCT bone.

Discussion:

Giant cell tumor (GCT) of the bone is a tumour of unknown histogenesist with distinct morphology. Some[3,14] categorise GCT bone into three grades, grade 1 (conventional tumour) Grade 2 (borderline tumour) and Grade three (malignant tumour). Mirra[15] believes that low-grade malignant neoplasm is an appropriate designation for GCT bone. Others[16 - 19] consider only two sub-types of the tumor, benign and malignant GCT boneon histology. The malignant GCT bone is a rare neoplasm[13]. It is also well known that histopathology alone may not always predict the behaviour of the tumour, since it is well recorded that histologically benignlooking GCT bone metastasized[20]. The molecular biology may help to settle this riddle in future.

The FNAC smears in the clinical and radio-graphic contxex are farely characteristic to suggests the diagnosis of GCT bone. Thus in present study 11 cases were correctly diagnosed as GCT. 1 case of GCT with secondary aneurismal bone cys was diagnosed as aneurysmal bone cyst and 1 case of primary malignant GCT as GCT conventional on FNAC, thus giving a sensitivity of 92.4% and specificity of 88.4 %. The case of GCT with ABC as wellas malignant GCT could have probably been sorted out with the help of radiologist and multiple punctures. Primary malignant cell is a rare tumor and therefore, not a big concern. There is a report of diagnosis of aggressive GCT on FNAC from sampling of soft tissues extention of the tumour [10]. FNAC diagnosis of GCT accuracy of 57 % to 91 % has been reported in the literature [6-8,13] GCT of the long bones, in variably arise from the epiphysis though rarely they may arise from the metaphysis as one case (case 13) in this study. In one study 3 out of 445 giant cell tumours arose from the metaphysis [18].

Before concluding the diagnosis GCT bone on FNAC smears. Tumors with giant cells and tumour like lesions need exclusion. These include Chondro-blastoma, Aneurysmal Bone Cyst, Brown Tumur of hype-parathyroidism, Giant cell tumur tendon sheath and Giant cell rich Osteo-sarcoma. In chontroblastoal smears are cellular with dispersed non-cohesive mono-nuclear cells having round to ovail nuclei, with occasional nuclear grooves. The giant cells are sparser and smaller.Chondroid matrix may be present in the back-ground. The cytology of Chondro-blastoma has been described by Fanning [21] and Kilpatrich [22]. Aneurysmalbone cyst yield frank blood with little cellular material of occasional osteo-clasts, osteo-blasts and fibrous strands. The Brown tumur smears show scattered mono-nuclear and giant cells. The Giant cell tumur tendon sheath is generally an extra-osseous lesion with dispersed mono-nuclear cells and giant cells. The mono-nuclear cells have scanty ill defined cytoplasm, with mono-morphic nuclei and irregular nuclear margins. The giant cells are dissociated from mono-nuclear cells, very scanty in number and may be small in size or in same size range as in GCT bone. Occasional collagen strands and macro-phages are seen (Fig 3). Giant cell rich Osteo-sarcoma show giant cells of Osteo-clast and/or malignant tumur giant cells, but clue to the diagnosis is the striking atypia and malighnat nature of the mon-nulcear tumour cells, irrespective of the presence or absence of osteoid(Fig 4). The clinical and radio-logic data, further help in differential diagnosis of all these lesions.

In summary, there-fore, the study amply demonstrates the utility of pre-operative FNAC diagnosis of GCT in the experienced hands with all advantages, including like simplicitly of procedure and least trauma to the patient, over open or needle core biopsy.

Table: FNAC-Giant Cell Tumor (Bone)7

S.No.	Age/Sex	Anat Site	FNAC	Histopath
01.	34/M	Upper tibia	GCT	GCT
02.	50/M	Femoral Condyle	GCT	GCT-Malignant
03.	20/M	Meta carpal	GCT	GCT
04.	25/M	Humerus(LE)	GCT	GCT
05.	19/F	Femur(LE)	GCT	GCT
06	17/F	Femur(LE)	ABC	GCT+ABC
07.	30/M	Femur(LE)	GCT	GCT
08.	35/F	Femur(LE)	GCT	GCT
09.	30/M	Humerus(UE)	GCT	GCT
10.	38/M	HumerusUE)	GCT	GCT
11(a).	35/F	Meta-carpals 4th & 5th	GCT	GCT
11(b)	---	Tumor recurrence	GCT (Conventional)	----
12.	17/M	Radius(LE)	GCT	GCT
13.	21/F	Femur (LE)	GCT	GCT

Abbreviations GCT- Giant Cell Tumour, UE— Upper End
LE— Lower End

References

1. Unni K. K: Dahlin Bone Tumours. General aspects and data on 11,087 cases. 5th Edition. 1994 Lippincott-Raven Philadelphi
2. Huros A G: Bone Tumors Diagonosis, treatment and prognosis. Philadelphia. W B. Saunders 1991 , pp 429 –467.
3. Schajowicz F. Tumors & tumor-like lesions of bone.Springer 2nd Edition : Springer, Berlin Heidelberg. New York.
4. Stromby N Akerman M : Cytodiagnosis of bone lesions by means of Fine needle aspiration biopsy. Acta Cytol 1973,17 ; 166 – 173.
5. EL Khoury GY,Terepka R H Mickelson M R : Fine needle aspiration biopsy of bone. J Bone Joint Surg. 1983,65a: 522 – 525.
6. Agarwal PK_Wahal KM : Cytopathologic study of primary tmors of bone & joints. Acta Cytol. 1983, 27: 23 – 27.
7. Seneige N. Ayala A, Carraso H.Mrray J. Raymond A. Giant cell tumor of bone. A Cytologic study of 24 cases. Diagn Cytopathology 1985 1 : 111 – 117.
8. Dollahite H Tatum L. Moinuddin SM. Carnesale PG. Aspiration biopsy of primary lesions of bone. J Bone Joint Surg. 1989. 71A: 1166 – 1169.
9. Vetrani A Fulciniti F. Boschi R. Marino G. Zeppa P . Troncone G. Palombini L : Fine needle aspiration diagnosis of giant cell tumor of bone. An experience with nine cases. Acta Cytol. 1990; 34: 863 – 867.
10. Rios-Martin J Otal- Salaverri C. Vazquez-Ramirez F. Ganzalez-Campora R. Davidson H. Fine needle aspiration diagnosis of aggressive giant tumor of bone. Acta Cytol. 1995 39: 650 – 654.
11. Kumar V . Rao C. Hazarika D Mukherjee G. Gowda B. Aspiration biopsy cytology of primary bone lesion. Acta Cytol 1993: 37(1). 83 – 89.
12. Jambhekar N Moonim M Khatib Y. Bone FNAC Expectation & Limitation. A Review (Part 1) J of Cytol 2001, 18 (1) 1 – 8.
13. Jambhekar N. Moonim M Khatib Y. Bone FNAC Expectation & limitation- A review (Part 11) J of Cytol 2001, 18 (20 57 – 72.
14. Goldenberg R R . Campbell C J Bonfiglio M. Giant cell tumor of bone : an analysis of 218 cases. J Bone Joint Surg. (Am) 1970. 52: 619 – 663.
15. Mirra JM. Bone tumors, diagnosis & treatment. J.B.Lippincott. Philadelphia. 1981.
16. Meyerding HW. Benign & malignant giant cell tumors of bone : Diagnosis & results of treatment . JAMA 1941 117: 1849 – 1855.
17. Jaffee HL Tumors & tumorous conditions of the bone & joints. Lea & Febiger, Philadelpline- 1958.
18. Dahlin Dc. Bone tumor Charles C Thomas, Springfield H. 1978.
19. Dahlin DC. Unni K K . Bone tumors .Bone tumors : general aspects & data on 8542 cases. Charles C Thombas Springfield H.1986.
20. Roek M Pritchard D Unik. Metastasis from histologically benign giant cell tumor of bone 1984. J Bone joint Surg. 66A; 269 – 274.
21. Fanning V, Sneige N,Canrasco C. Ayala A. Marray J. Raymound A. Fine needle aspiration cytology of Chondroblastoma Caner 1990, 65: 1847 – 1863.
22. Kilpatrick S. Pik E, Geisinger K. Ward W .Chondroblastoma of bone. Use of fine needle aspiration biopsy & potential diagnosic pitafalls. Diagn Cytolpath 1997 : 16 : 65 – 71.

SARS: FIRST GLOBAL EPIDEMIC OF 21ST. CENTURY

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Throughout history human populations have undergone shifts in their relations with disease - shifts that are always linked to major changes in the way people interact with the environment. There have been three epidemiological transitions. The first epidemiological transition took place 10,000 years ago, when people abandoned their nomadic existence and began farming. That profoundly new way for life disrupted ecosystems and created denser living conditions that led to new diseases. The second epidemiological transition was during middle of the 19th. century when the industrial revolution took hold in the USA and Europe (giving rise to diseases of civilization: cancer, clogged arteries, diabetes, osteoporosis), particularly during 1971 when the war against infectious diseases seemed to have been won. And in the past two decades the emergence of illnesses such as hepatitis C, cat scratch disease, Ebola virus, Hanta virus and others has created a third epidemiological transition, a disheartening set of changes that in many ways have reversed the effects of the second transition and coincide with the shift to globalism. Burgeoning population growth and urbanization, widespread environmental degradation, including global warming and tropical deforestation, and radically improved methods of transportation have given rise to new ways of contracting and spreading disease.

People and pathogens have a long history together. Infections have been detected in the bones of human ancestors more than a million years old, and the evidence from the mummy of the Egyptian pharaoh Ramses V suggests that he may have died from smallpox more than 3,000 years ago. Widespread outbreaks of disease are also well documented. Between 1347 and 1351 roughly a third of the population of medieval Europe was wiped out by bubonic plague, which is carried by fleas that live on rodents. In 1793, 10 percent of the population of Philadelphia succumbed to yellow fever, which is spread by mosquitoes. And in 1875 the son of the Fiji chief came down with measles after a ceremonial trip to Australia. Within four months more than 20,000 Fijians were dead from the imported disease, which spreads through the air when its victims cough or sneeze.

People and invading microorganisms evolve together: people gradually become more resistant, and the micro organisms become less virulent. The result is either mutualism, in which the relation benefits both species, or commonsalism, in which one species benefits without harming the other. Chicken pox and measles, once fatal afflictions, now exist in more benign forms. The best interests of an organism are not served if it kills its host; doing so would be like picking a fight with the person who signs your paycheck.

We are living in the twilight of the antibiotic era. Within our lifetimes, scraped knees and cut fingers may return to the realm of fatal conditions. The third epidemiological transition is restoring a more traditional relationship between human and beastie. We may be returning to pre-antibiotic circumstances more familiar to our ancestors than to us, in which sudden, unpredictable and often fatal illness was a way of life.

The modern world is becoming a viral superhighway. Our newly global society is characterized by huge increases in population, international travel and trade - factors that enable diseases to spread much more readily than ever before from person to person and from continent to continent. By 2020 the world population will have surpassed seven billion, and half those people will be living in urban areas. Developing countries are already hard-pressed to provide severs, plumbing and other infrastructure; in the future, clean water and adequate sanitation could become increasingly rare. Meanwhile, political upheavals regularly cause millions of people to flee their homelands and gather in refugee camps, which become Petri dishes for germs.

Left unchecked, today's emerging diseases can become the endemic diseases of tomorrow. This is what happened with HIV/AIDS, which spread from a remote part of Africa to all other continents 20 years ago, and is now entrenched all over the world, necessitating a major international control efforts.

We humans are the self-proclaimed smartest creatures on the planet. But no matter how scientific or advanced we become, we are still at the mercy of Nature. What better proof do you want of that than the fact we have not been able to find a cure for the commonest of all ailments, common cold? SARS now holds humans to ransom. The entire world has become its target. Imagine if those billions of dollars wasted on Iraq war were diverted to finding a cure for SARS!

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Global epidemic of 21st. century

SARS is the first global epidemic of the 21st Century. It will historically be seen that way. Nature, it appears, is reacting to human population growth by evolving diseases faster than we can keep up: these new pandemics may simply be a natural population control mechanism.

SARS cases continue to be reported from a growing number of countries. A cumulative total of 8295 cases with 750 deaths (as on May 29,2003) have been reported from 33 countries. SARS was first identified in Viet Nam on 28 February, when Dr Carlo Urbani, an epidemiologist from the WHO office in Hanoi, examined a patient with a severe form of pneumonia with no known cause. By 11 March, at least 20 hospital workers in Hanoi's private French Hospital, and 23 at a hospital in Hong Kong, were ill with a similar acute respiratory syndrome.

SARS is the first severe and readily transmissible new disease to emerge in the 21st century. Though much about the disease remains poorly understood and frankly puzzling, SARS has shown a clear capacity for spread along the routes of international air travel. At present, the outbreaks of greatest concern are concentrated in transportation hubs or spreading in densely populated areas. WHO regards every country with an international airport, or bordering an area having recent local transmission, as at potential risk of an outbreak. The first cases of SARS are now known to have emerged in mid-November 2002 in Guangdong Province, China. The first official report of an outbreak of atypical pneumonia in the province, said to have affected 305 persons and caused 5 deaths, was received by WHO on 11 February. Around 30% of cases were reported to occur in health care workers. In the meantime, SARS was carried out of Guangdong Province on 21February by an infected medical doctor who had treated patients in his home town. He brought the virus to the ninth floor of a four-star hotel in Hong Kong. Days later, guests and visitors to the hotel's ninth floor had seeded outbreaks of cases in the hospital systems of Hong Kong, Viet Nam, and Singapore. Simultaneously, the disease began spreading around the world along international air travel routes as guests at the hotel flew home to Toronto and elsewhere, and as other medical doctors who had treated the earliest cases in Viet Nam and Singapore traveled internationally for medical or other reasons. When the disease moved out of southern China, the outbreaks it seeded – in Hanoi, Hong Kong, Singapore, and Toronto – became the initial "hot zones" of SARS, characterized by rapid increases in the number of cases, especially in health care workers and their close contacts. In these areas, SARS first took root in hospital settings, where staff, unaware that a new disease had surfaced and fighting to save the lives of patients, exposed themselves to the infectious agent without barrier protection. All of these initial outbreaks were subsequently characterized by chains of secondary transmission outside the health care environment. Epidemiological analysis indicated that the new disease was spreading along the routes of international air travel. WHO immediately issued emergency travel recommendations to alert health authorities, physicians, and the travelling public to what was now perceived to be a worldwide threat to health. The global alert achieved its purpose. After the recommendations, all countries with imported cases, with the exception of provinces in China, were able, through prompt detection of cases, immediate isolation, strict infection control, and vigorous contact tracing, to either prevent further transmission or keep the number of additional cases very low. During the last week of April, the outbreaks in Hanoi, Hong Kong, Singapore, and Toronto showed some signs of peaking. On 28 April, Viet Nam became the first country to stop local transmission of SARS. However, new probable cases, including cases in hospital staff, additional deaths, and first cases imported to new areas continued to be reported from several countries.

SARS demonstrates dramatically the global havoc that can be wreaked by a newly emerging infectious disease. At this moment, public health authorities, doctors, nurses, scientists, and laboratory staff around the world are struggling to cope with SARS at a time when some hope remains that the disease might still be contained. Economists and market analysts are simultaneously struggling to calculate the present and future costs, initially estimated at US$ 30 billion in the Far East alone. Public panic is widespread, some government officials have lost their jobs, and social stability has been jeopardized in some of the hardest hit areas. Hospitals, schools, and borders have been closed, and several governments have advised their citizens not to travel to hard-hit areas.

SARS needs to be regarded as a particularly serious threat for several reasons. The disease has no vaccine and no treatment, forcing health authorities to resort to control tools dating back to the earliest days of empirical microbiology: isolation and quarantine. The virus comes from a family notorious for its frequent mutations, raising important questions about the future evolution of outbreaks and prospects for vaccine development. Epidemiology and pathogenesis are poorly understood. The initial symptoms are non-specific and common. All available diagnostic tests have important limitations. If tests are poorly conducted or results wrongly applied, patients excreting virus and thus capable of infecting others can slip through the safety net of isolation and infection control. The disease continues to show a disturbing concentration in previously healthy hospital staff – the human resource vital to control. A significant proportion of patients require intensive care, thus adding to the considerable strain on hospital and health care systems. Evidence is mounting that certain source cases make a special contribution to rapid spread of infection. The maximum incubation period, currently estimated at 10 days, allows spread via air travel between any two cities in the world. WHO's most recent analysis estimates overall case fatality in the range of 14% to 15%. In persons over the age of 65, the case fatality ratio can exceed 50%.

AIDS and SARS have some features in common: resistance to antibiotics and other drugs, species-jumping nature, that is, they may be harmless in one species but fatal in another (a good example is Creutzfeldtz Jacob Disease, which is harmless in sheep) and result of human interaction with animals (there is a theory that the AIDS virus crossed over into humans through blood letting ceremonies involving the African green monkey).

Epidemiological profile

The primary way that SARS appears to spread is by close person-to-person contact. Most cases of SARS have involved people who cared for or lived with someone with SARS, or had direct contact with infectious material (for example, respiratory secretions) from a person who has SARS. Potential ways in which SARS can be spread include touching the skin of other people or objects that are contaminated with infectious droplets and then touching your eye(s), nose, or mouth. This can happen when someone who is sick with SARS coughs or sneezes droplets onto themselves, other people, or nearby surfaces. It also is possible that SARS can be spread more broadly through the air or by other ways that are currently not known.

Workers, who in the last 10 days have traveled to a known SARS area, or have had close contact with a co-worker or family member with suspected or probable SARS could be at increased risk of developing SARS and should be vigilant for the development of fever (greater than 100.4° F) or respiratory symptoms (e.g., cough or difficulty breathing). If these symptoms develop you should not go to work, school, or other public areas but should seek evaluation by a health-care provider and practice infection control precautions recommended for the home or residential setting; be sure to contact your health-care provider beforehand to let them know you may have been exposed to SARS so arrangements can be made, as necessary, to prevent transmission to others in the healthcare setting.

The virus can survive as long as 24 hours outside the body: doctors and nurses who touch their protective gear while changing into regular clothing may be unwittingly exposing themselves and others to the coronavirus.

The risk of death from SARS may have less to do with a particular strain of the virus and more to do with the body's reaction to it. The immunological and inflammatory response of the body could be contributing significantly to the damage in the lungs. But nailing that down, along with questions of whether survivors become immune to further infection and whether the disease is permanently with us, like AIDS, will take more research.

SARS has been associated etiologically with a novel coronavirus, SARS-CoV. Evidence of SARS-CoV infection has been identified in patients with SARS in several countries, including the United States. Several new laboratory tests can be used to detect SARS-CoV. Serologic testing for coronavirus antibody can be performed by using indirect fluorescent antibody or enzyme-linked immunosorbent assays that are specific for antibody produced after infection. Although some patients have detectable coronavirus antibody during the acute phase (i.e., within 14 days of illness onset), definitive interpretation of negative coronavirus antibody tests is possible only for specimens obtained >21 days after onset of symptoms. A reverse transcriptase polymerase chain reaction (RT-PCR) test specific for viral RNA has been positive within the first 10 days after onset of fever in specimens from some SARS patients, but the duration of detectable viremia or viral shedding is unknown. RT-PCR testing can detect SARS-CoV in clinical specimens, including serum, stool, and nasal secretions. Finally, viral culture and isolation have both been used to detect SARS-CoV. Absence of SARS-CoV antibody in serum obtained <21 days after illness onset, a negative PCR test, or a negative viral culture does not exclude coronavirus infection.

Reported U.S. cases of SARS still will be classified as suspect or probable; however, these cases can be further classified as laboratory-confirmed or -negative if laboratory data are available and complete, or as laboratory-indeterminate if specimens are not available or testing is incomplete. Obtaining convalescent serum samples to make a final determination about infection with SARS-CoV is critical.

The Centers for Disease Control and Prevention (CDC) has sequenced the genome for the coronavirus believed to be responsible for the global epidemic of severe acute respiratory syndrome or SARS. The sequence data confirm that the SARS coronavirus is a previously unrecognized coronavirus. The availability of the sequence data will have an immediate impact on efforts to develop new and rapid diagnostic tests, antiviral agents and vaccines. This sequence information will also facilitate studies to explore the pathogenesis of this new coronavirus. All of the sequence, except for the leader sequence, was derived directly from viral RNA. The genome of the SARS coronavirus is 29,727 nucleotides in length and the genome organization is similar to that of other coronaviruses. Open reading frames corresponding to the predicted polymerase protein (polymerase 1a, 1b), spike protein (S), small membrane protein (E), membrane protein (M) and nucleocapsid protein (N) have been identified. Early in the course of disease, the absolute lymphocyte count is often decreased. Overall white cell counts have generally been normal or decreased. At the peak of the respiratory illness, up to half of patients have leukopenia and thrombocytopenia or low-normal platelet counts (50,000 – 150,000 / µl). Early in the respiratory phase, elevated creatine phosphokinase levels (up to 3000 IU / L) and hepatic transaminases (2- to 6-times the upper limits of normal) have been noted. Renal function has remained normal in the majority of patients.

Clinical Description

The surveillance case definitions based on available clinical and epidemiological data are now being supplemented by a number of laboratory tests and will continue to be reviewed as tests currently used in research settings become more widely available as diagnostic tests.

Suspect case

1. A person presenting after 1 November 2002[1] with history of:

- high fever (>38 °C)

AND

- cough or breathing difficulty

AND one or more of the following exposures during the 10 days prior to onset of symptoms:

- ***close contact*** with a person who is a suspect or probable case of SARS;
- history of travel, to an area with recent local transmission of SARS
- residing in an area with recent local transmission of SARS

2. A person with an unexplained acute respiratory illness resulting in death after 1 November 2002, but on whom no autopsy has been performed

AND one or more of the following exposures during to 10 days prior to onset of symptoms:

- ***close contact,*** with a person who is a suspect or probable case of SARS;
- history of travel to an area with recent local transmission of SARS
- residing in an area with recent local transmission of SARS

Probable case

1. A suspect case with radiographic evidence of infiltrates consistent with pneumonia or respiratory distress syndrome (RDS) on chest X-ray (CXR).
2. A suspect case of SARS that is positive for SARS coronavirus by one or more assays.
3. A suspect case with autopsy findings consistent with the pathology of RDS without an identifiable cause.

Exclusion criteria

A case should be excluded if an alternative diagnosis can fully explain their illness.

Initial diagnostic testing for suspected SARS patients should include chest radiograph, pulse oximetry, blood cultures, sputum Gram's stain and culture, and testing for viral respiratory pathogens, notably influenza A and B and respiratory syncytial virus. A specimen for Legionella and pneumococcal urinary antigen testing should also be considered. Clinicians should save any available clinical specimens (respiratory, blood, and serum) for additional testing until a specific diagnosis is made. Acute and convalescent (greater than 21 days after onset of symptoms) serum samples should be collected from each patient who meets the SARS case definition.

Clinicians evaluating suspected cases should use standard precautions (e.g., hand hygiene) together with airborne (e.g., N-95 respirator) and contact (e.g., gowns and gloves) precautions. Until the mode of transmission has been defined more precisely, eye protection also should be worn for all patient contact.

Treatment regimens have included a variety of antibiotics to presumptively treat known bacterial agents of atypical pneumonia. In several locations, therapy has also included antiviral agents such as oseltamivir or ribavirin. Steroids have also been given orally or intravenously to patients in combination with ribavirin and other antimicrobials. At present, the most efficacious treatment regime, if any is unknown.

Laboratory testing

Researchers in several countries are working towards developing fast and accurate laboratory tests for the SARS. However, until those tests have been adequately field tested and shown to be reliable, SARS diagnosis remains dependant on the clinical findings of an atypical pneumonia not attributed to another cause and a history of exposure to a suspect or probable case of SARS or their respiratory secretions and other bodily fluids.

Antibody tests - ELISA (Enzyme Linked Immuno-Sorbant Assay) detects antibodies in the serum of SARS patients reliably as from day 21 after the onset of clinical symptoms and signs.

Immunofluorescence Assays detect antibodies in serum of SARS patients after about day 10 of illness onset. This is a reliable test requiring the use of fixed SARS-virus, an immunofluorescence microscope and an experienced microbiologist. Positive antibody tests indicate that the patient was infected with the SARS -virus.

Molecular tests (PCR) - can detect genetic material of the SARS -virus in various specimens (blood, stool, respiratory secretions or body tissue). Primers, which are the key pieces for a PCR test, have since been used by numerous countries around the world. A ready-to-use PCR test kit containing primers and positive and negative control has been developed. Testing of the kit by network members is expected to quickly yield the data needed to assess the test's performance, in comparison with primers developed by other WHO network laboratories. Existing PCR tests are very specific but lack sensitivity. That means that negative tests can't rule out the presence of the SARS virus in patients. Various WHO network laboratories are working on their PCR protocols and primers to improve their reliability.

Cell culture - Virus in specimens (such as respiratory secretions, blood or stool) from SARS patients can also be detected by infecting cell cultures and growing the virus. Once isolated, the virus must be identified as the SARS virus with further tests. Cell culture is a very demanding test, but the only means to show the existence of a live virus.

Hospital Infection Control

Those presenting to health care facilities who require assessment for SARS should be rapidly diverted by triage nurses to a separate area to minimize transmission to others. Those patients should be given a N 95 mask to wear. Staff involved in the triage process should wear a N 95 mask and wash hands before and after contact with any patient, after activities likely to cause contamination and after removing gloves. Wherever possible, patients under investigation for SARS should be separated from the probable cases. SARS cases should be isolated and accommodated in descending order of preference as: negative pressure rooms with the door closed, single rooms with their own bathroom facilities, cohort placement in an area with an independent air supply, exhaust system and bathroom facilities. Strict adherence to the barrier nursing of patients with SARS, using precautions for airborne, droplet and contact transmission is essential. Disposable equipment should be used wherever possible in the treatment and care of patients with SARS and disposed of appropriately. If devices are to be reused, they should be sterilized in accordance with manufacturers' instructions. Surfaces should be cleaned with broad spectrum disinfectants of proven antiviral activity. Movement of patients outside of the isolation unit should be avoided. If moved the patients should wear a N 95 mask. Hand washing is crucial. PPE (personal protective equipment) should be worn by all staff and visitors accessing the isolation unit. The PPE should include N95 mask as a minimum, single pair of gloves, goggles, disposable gown, apron, and footwear that can be decontaminated. All sharps should be dealt with promptly and safely. Linen from the patients should be prepared on site for the laundry staff. When probable SARS cases are promptly detected, isolated, and managed according to strict procedures of infection control, further spread to hospital staff and family members either does not occur at all or results in a very small number of secondary infections. The SARS virus spreads, in the vast majority of cases, through exposure to respiratory droplets during close face-to-face contact. Because the virus is new, much about its behaviour is poorly understood. Scientists are working to determine the amount of time the virus can survive in the environment on both dry surfaces and in suspension, including in faecal matter.

Responding to challenge

The management of SARS requires a proactive and planned approach to ensure the appropriate prevention and control of the spread of disease by health authorities. In the absence of effective treatment modalities and non-availability of vaccine, prevention is an absolute necessity.

Careful attention to infection-control precautions, both in home and health-care settings, remains critical to containment of SARS. Symptomatic persons should use infection-control precautions to minimize the potential for transmission and should seek health-care evaluation. Patients should inform health-care providers about the symptoms in advance so arrangements can be made, if necessary, to prevent potential transmission to others in the health-care setting. Patients in ambulatory settings should be screened promptly for fever, respiratory symptoms, recent travel, and close contact with SARS patients.

Future threat

Since this strain is relatively new to humans, genetic mutations which improve its ability to survive in our cells are possible. You only get one chance to eradicate something like this - once it's established, you've got a real problem. If

future genetic mutations make SARS more transmissible, the world would have a far bigger problem.
We need to get access to information about disease outbreaks as soon as they occur and to share that information globally even faster than modern travel. We need to ensure this remains the case, so that SARS does not become a burden on countries least able to afford it. We also need to share successful strategies to contain the disease and learn from what does not work. Effective surveillance and rapid response save lives, protects economies and is an essential pillar of both national and international security.

The way forward

The disease could be stopped if everyone worked together. What is important is for all countries to participate and to help prevent it getting in a place where it would be very difficult to stop it. Let's equip our scientists with money and everything they need in this fight. After all, it's our lives they are fighting for.

The world still had a window of opportunity to avoid the virus becoming endemic, such as flu and HIV... to contain it - lessen it where it is, and stop it spreading. The WHO says Vietnam - which has reported no new cases of SARS in 18 days - could be declared the first country to have contained its outbreak. SARS can still be brought under control - and perhaps even eradicated. It appears to be contained, certainly in developed countries, by very good containment and monitoring practices. The best way to tackle it is for governments to act swiftly and forcefully to isolate it. Let's join hands and win this SARS war first - for ourselves and for posterity. We must be vigilant. The battle is not yet won. Our strategy should be to do what is possible to defeat SARS. We should not conclude that we will just have to accept it like all other diseases already affecting humankind. Globalization of disease and threats to health mean globalization of the fight against them.

References

1. Armelagos G J. The Viral Superhighway. The Sciences. January 1998: 24-29.
2. Amin Tabish. Emergent Infection: A Global Health Threat. Ann of NAMS. 1998; 34(4): 235-248
3. Amin Tabish. Tackling Emerging Infectious Diseases. J Med Sci. 1999; 2(2): 12-16.
4. World Health Organisation. Cumulative Number of Reported Probable Cases of Severe Acute Respiratory Syndrome (SARS). From: 1 Nov 2002[1] to: 29 May 2003
5. Ksiazek TG, Erdman D, Goldsmith C, et al. A novel coronavirus associated with severe acute respiratory syndrome. N Engl J Med. Drosten C, Gunther S, Preiser W, et al. Identification of a novel coronavirus in patients with severe acute respiratory syndrome. N Engl J Med.
6. CDC. Update. Severe Acute Respiratory Distress Syndrome. SARS Coronavirus Sequencing, April 24, 2003.

VIRTUAL RADIOLOGY AND ITS IMPLICATIONS IN SMHS HOSPITAL.

Bakshi Jehangir MD ; Asifa Nazir MBBS

Introduction

The emergence of computerised imaging modalities, such as computed tomography (CT) and magnetic resonance imaging (MRI) have initiated the digital age of radiology. Recently, even traditional analog or hard-copy formats, such as plain film X-rays and mammography, have begun to be acquired or processed into digital format which is a filmless or virtual format (digital radiography). This has permitted the development of filmless or virtual radiology departments in which all images are acquired and stored digitally by computers[1]. The use of high-speed telecommunication has permitted geographically separated health centres to access images at workstations around the world, forming a virtual radiology network.

Virtual Radiology System

The core of a virtual radiology system is the ***Picture Archiving and Communication System*** (PACS). **PACS** is a system that permits the acquisition, storage, retrieval and display of digital images and also facilitates the communication of these images over a ***Local Area Network*** (LAN) connection[2]. The end-user computer software through which images are accessed, transmitted and viewed is called a ***DICOM server***. This software is capable of receiving and transmitting radiological images from all imaging modalities connected to the virtual radiology system and features tools for image display and manipulation, measurement, interactive window leveling, cross referencing, and study comparison. It operates on the ***DICOM-3*** (Digital Imaging and Communications in Medicine version 3) standard, the international standard for teleradiology, which ensures compatibility between different centres that may employ different forms of technology in their respective radiology departments.

In this system, a radiological image is first captured in a digital format (obtained digitally or via the conversion of an analog film to a digital format), and then sent to the server which not only allows the transmission and viewing of images over a network but also permits a number of technical manipulations. A copy of each image can also be simultaneously sent to an archive server and it is stored permanently within a library of **Compact Discs** (CD) or **Digital Library Tapes** (DLT).

Through the process of teleradiology, any image can be accessed from any on-line workstation within the local area network (LAN) at a transfer rate of 100 Megabits per second. It can also enable the communication of digital images between the LAN and outside locations to users who have been granted offsite access to the PACS (such as radiologists reading radiographs from their PCs) over existing telephone lines (POTS) or the Internet.

Advantages of virtual radiography

- ☐ Digital radiographs have improved viewing quality.
- ☐ Computers allow for easier sorting and viewing of multiple images which can quickly be located and accessed for reporting or teaching purposes.
- ☐ Digital images are immediately accessible and can be viewed after-hours and at remote locations. In addition, when radiographs are combined with electronic patient charts, the differential diagnosis can be narrowed, thus increasing the speed of clinical decision making[1].
- ☐ The ability to manipulate and enhance these digital images allows the radiologist to extract maximum information from an image during the initial examination. Post-acquisition manipulation of digital pictures using advanced technology improves the radiologist's ability to detect abnormalities[4].
- ☐ Educational programs that include radiological images and associated case histories that can be used by students to learn radiology[1,5]. For example, 3D digital imaging programs have been designed to aid students in gaining orientation about the radiographic presentation of human anatomy.
- ☐ Digital radiographic images require less physical storage space than traditional films and can be stored with patients computer records for future reference. Storage on disk decreases the chance of losing images, which often occurs with films[4].
- ☐ Picture archives of radiological images have been developed and placed on the Internet where they

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can be accessed by users around the world[6].The ability to place digital images on the World Wide Web thus serves as an excellent source for continuing medical education[7].

Implications of virtual radiology in SMHS Hospital

SMHS Hospital provides secondary, tertiary and quaternary medical care in all allied specialities and super-specialities of Medicine and Surgery to the people of Kashmir and is the main Associated Teaching Hospital of Government Medical College, Srinagar for imparting undergraduate and postgraduate medical training.

The development of a virtual radiology system at SMHS Hospital has many potential advantages:

- ☐ Since the modalities of computed tomography (CT) and magnetic resonance imaging (MRI) installed at the Diagnostic Imaging Research Centre of SMHS Hospital have been sourced from different vendors, the setting up of a local virtual radiology network can allow centralised viewing and reporting of these investigations.
- ☐ Radiological images and reports from these modalities as well as from digitised analog radiographs can be promptly transmitted to remote computers installed in the various departments of the hospital and also to the other associated hospitals of Government Medical college, Srinagar.
- ☐ Large numbers of radiological images can be stored with relevant patient data in a PACS in the form of a virtual library and can easily be located and accessed for reporting or teaching purposes. Using inexpensive and readily available software a digitised teaching collection can be created from hard-copy films already existing in the library of the Diagnostic Imaging Research Centre of SMHS Hospital[8].
- ☐ A pioneering basic Teleradiology project using the Internet to discuss Spiral CT and MRI images from the Diagnostic Imaging Research Centre of SMHS Hospital with radiologists from western countries is currently underway[9].

Conclusion

Filmless or virtual radiology is relatively uncommon in India and almost non-existent in Kashmir. The use of radiographic film is gradually being replaced by digital imaging and the Picture Archiving and Communication System (PACS)[10-14]. Given the importance of SMHS Hospital as a primary site of medical care and education in Kashmir, the setting up of a virtual radiology system can yield immediate benefits as well as help in keeping up with future advances in technology.-

References

1. Goldberg REA, Borissova D, and Penner A. The virtual radiology department: Basic concepts, considerations, and implications. Current Oncology (1998) ; 6 : 24-31.
2. Owen CJ, Champagne JP, Aggarwal A and Bitar R. Virtual Radiology - Digital imaging takes on new importance at the University Health Network (UHN) Univ Toronto Med J (2000); 77 : 178-80.
3. Hynes D, Stevenson G, and Nahmias C. Towards filmless and distance radiology. The Lancet (1997) ; 350 : 657-660.
4. Goodman TR and McHugh K. Advances in radiology. Arch Dis Child (1997) : 77 : 265-271.
5. Kuszyk BS, Calhoun PS, Soyer PA, and Fishman EK. Computers in radiology: An interactive computer-based tool for teaching the segmental anatomy of the liver: Usefulness in education of residents and fellows. AJR (1997) ; 169 : 631-634.
6. Richardson ML. Computers in radiologic education: A world wide web radiology teaching file server on the Internet. AJR (1995) ; 164 : 479-483.
7. McEnery KW. Computers in radiologic education: The Internet, world wide web, and mosaic: An overview. AJR (1995) ; 164 : 469-473.
8. Mayo-Smith WW, Ngan K, Hou D, Gupta H, Dupuy DE and Abbott GF. Digitizing your hard-copy teaching file with inexpensive, commercially available software. Acad Radiol (2001); 8 : 273-277.
9. Jehangir B. and Nazir A. Telemedicine - Doctors without borders. JK-Practitioner(2003);10(2):158-159
10. Watkins J. A hospital-wide picture archiving and communication system (PACS): the views of users and providers of the radiology service at Hammersmith Hospital. Eur J Radiol (1999); 32 :106-112.
11. Honeyman JC. Information systems integration in radiology. J Digit Imaging (1999) ; 12 : 218-222.
12. Jones WG. Crossing the digital divide: moving from film to filmless radiology. J Digit Imaging (1999) ; 12 : 47-49.
13. Margulis AR, Sunshine JH. Radiology at the turn of the millennium. Radiology (2000) ; 214 : 15-23.
14. Pilling JR. Lessons learned from a whole hospital PACS installation. Clin Radiol (2002) ; 57 :784-88.

MICRONUTRIENTS

G.M. Malik

When we think of providing the body with "nutrient help" the term

"SUPPLEMENTS" pops in to consciousness...

Supplements is the popular word used to refer to micro-nutrients such as vitamins, minerals, enzymes, probiotics, and even hormones.

Micro-nutrients are essential to health... to have and keep health ,we must consume a variety of wholefoods raised in organic rich soil so that the foods raised will contain the full spectrum of macro and micro nutrients... Unfortunately, for a variety of reason, often macro-nutrients (protein, carbohydrates, fats) do not contain sufficient micro-nutrients for optimum health and longevity, thus it is wise to consume supplements in the form of superfoods and/or concentrated vitamins and minerals extracts from food suurces.

What are Micronutients?

Micronutrients are vitamins and minerals that are needed by the body in very small ammounts but are essential for the body to maintain its normal functions. Without them, the body can not function optimally and different health problems occur. As they can not be synthesized in the body, they must be provided by the diet. If these micronutrients are missing during phase of rapid growth, the development of basic biological function like intellect, and even life itself can be threatened. This is why young children and pregnant women are often among the risk groups for micronutrient deficiencies.

Iron Deficiency Anaemia

Iron Deficiency Anaemia occurs at that stage when body specially requires more quantity of Iron elements:

- During pregnancy-when fetus develops in womb and development of mothers tissues also take place.
- During adolescence-the body develops very fast and lot of blood waste due to menstruation.
- Among pre- School children-when over all growth happens at very fast pace.

Iron Defiency Anaemia (IDA) / Nurtitional Anaemia

During pregnancy:

- Increases incidence of maternal deaths by 20%
- Increases risk of premature deliveries and low birth weight (LBW*) babies
- Increases neonatal mortality
- Contributes to irreversible brain damage

in children:

- Reduces physical stamina
- Lowers IQ by 5-10 points
- Contributes to poor cognitive development
- Lower attention and concentration span leading to poor school performances
- Increase susceptibility to infections

in adults:

- Reduces capacity to do physical work
- Reduces productivity
- Increase susceptibility to infections.

Prevalience of Anemia Among Children (Aged 6-35 Months

In India approximately three—quarters of children age 6-35 months (74.3 percent) are anemic, including a large majority of children in every subgroup of the population. The prevalence of anemia is particularly high among children who are one year old, boys, and children living in households with a low to medium standard of living. Children's whose mothers are anemic are more likely to be anemic themselves than are other children.

Maternal Anemia

Overall, 52 percent of women in India have some degree of anemia. Twenty percent of women in country are moderately to severely anemic, compared with 15 percent of women in Uttar Pradesh. Anemia is a serious problem among women. Pregnant women are much more likely than non-pregnant women to be modertely to severely anemic.

Vitamin A Defiency (VAD)

Vitamin 'A' deficiency is especially common among the children aged 6-36 months from lower income group residing in hilly areas. It is a very effective health problem of India.

Vitamin A Defiency (VAD)

During pregnancy:

- Results in low stores of vitamin A in the new-born
- Reduces breast milk content of vitamin A
- Contributes to maternal mortality.

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In Children

- Causes eye disorders ranging from night blindness to permanent blindness.
- Increase risk of diarrhea by three times.
- Doubles the risk of acute respiratory infections (ARI)
- Increases risk of death by 6 to 9 times.
- Increases duration ad severity of diarrhea and measles.

According to Natioonal Family Health Survey-2 1998-99, in India only 29.7 percent children received at least one dose of Vitamin 'A' supplementation. Proportion of those children who received any one dose during last 6 months was only 17 percent. Accordig to Reproductive and Child Health (RCH)- Rapid Household Survey in entire India only one-third children receive at least one dose of Vitamin 'A'.

Iodine Defiency Disorders (IDD)

Iodine is a micro-nutrient essential to human life. The average daily requirement of an adult is 150 micrograms. About 90 percent of this normally come from food, and the rest from water. Food crops and water derive iodine from the soil. Consumption of crops and plants from iodine deficient soils, leads to iodine deficiency in population. The wide spectrum of disorders caused by inadequate iodine consumption are known collectively as iodine deficiency disrders (IDD). No state in India is free from IDD.

Deficiency of iodine is pregnant women can cause irreversible brain damage in the unborn baby. People living in areas affected by severe IDD may have an intelligence quotient (IQ) of up to about 13.5 points below that of those from comparable communities in areas where there is no iodine deficiency. This mental deficiency has an immediate effect on child learning capacity, women's health, the quality of life of communities, and economic productivity.

Iodine Defiency Disorders (IDD)

During pregnancy:

- Causes spontaneous abortions stillbirths and infant deaths.
- Interferes with brain development of the foetus
- Attributes to birth of low IQ babies.

In Children:

- Lowers IQ by as many as 10-15 points.
- Impairs mental and cognitive function, leading to poor school performance
- Retards growth
- Dulls motivation and curiosity; reduces play and exploratory activites.
- Results in muscular disorders, speech and hearing defects, impaired coordination and sluggishness.

Only 49.4% of the households in India use iodized salt at the recommended level of 15 parts per million (ppm) suggesting that IDD is likely to be a serious problem in the state. Rural households and households with a low standard of living are much less likely than other households in Uttranchal to use iodized salt.

The MOST Mission

The MOST mission is to 1) maintain and enhance USAID's global leadership position in addressing micronutrient malnutrition, particularly vitamin A deficiency; 2) implement and evaluate state-of-the-art interventions to alleviate micronutrient deficiencies; and 3) provide technical guidance and coordination to other USAID projects with micronutrient - related components.

The MOST Strategy

The MOST strategy is built upon a framework of global and country-level results:

The global agenda focuses on 1) promoting a revised global agenda in collaboration with other organizations worldwide committed to reducing micronutrient malnutrition; 2) translating scientific knowledge into policy and program action; and 3) maximizing lessions learned through USAID's extensive field program experience.

Country-level results address deficiecies in vitamin A, iron, and iodine: 1) vitamin A coverage of at least 80 percent of deficient children under 6 years of age; 2) moderate to severe anemia decreased by 30 percent in pregnant women and children 6-24 months of age; and 3) percentage of the population with symptoms of iodine deficiency reduced by 30 percent.

Key Areas of Activity

- Application of behavior change techniques to create demand for micronutrient programs and services
- Enhancement of the effectiveness and sustainability of supplementation programs.
- Sound planning, implementation and quality control of fortification programs.
- Inclusion of other food-based approaches in programs.
- Application of appropriate economic analysis to guide the evolution of country programs.
- Use of monitoring and evaluation to improve program operations.
- Development of public and private sector alliances to enhance the effectiveness of interventions.

Target Groups

MOST focuses on the improvement of the micronutrient status of children under 6 years of age and women of childbearing age. Several intervention options available to address micronutrient dificiency, such as food fortification, will benefit not only those target groups, but also school-age children and adult males.

ACHALASIA CARDIA - A RARE OESOPHAGEAL MOTILITY DISORDER

Achalasia cardia is a rare, most easily recognized and best defined motor disorder of esophagus. It affects both sexes equally and usually presents in adult life. Achalasia is characterized by I) failure of complte relaxation of lower esophageal sphincter (LES) to swallowing II) aperistalsis in smooth muscle esophagus. The resting LES pressure is raised in about 60% of cases.

Clinical manifestations include dysphagia, regurgitation, chest pain, weight pain, weight loss and aspiration pneumonia.

A barium swallow or esophageal manometry can demonstrate the physiological abnormalities of achalasia. the characteristic x-ray is of a dilated intrathoracic esophagus with impaired emptying, an air fluid level, absence of a gastric air bubble and an LES that that tapers to a point giving the distal esophagus a beak like appearance.

Treatment of achalsia is directed at compensating for the functional abnormalities and preventing complications because the underlying neuropathy can't be corrected. The main of aim of treatment is to reduce the LES pressure and with reduced LES pressure gravity promotes esophageal emptying. LES pressure can be reduced by pharmacological therapy, ballon dilatation, surgical myotomy Botolinum toxin injection of LES.

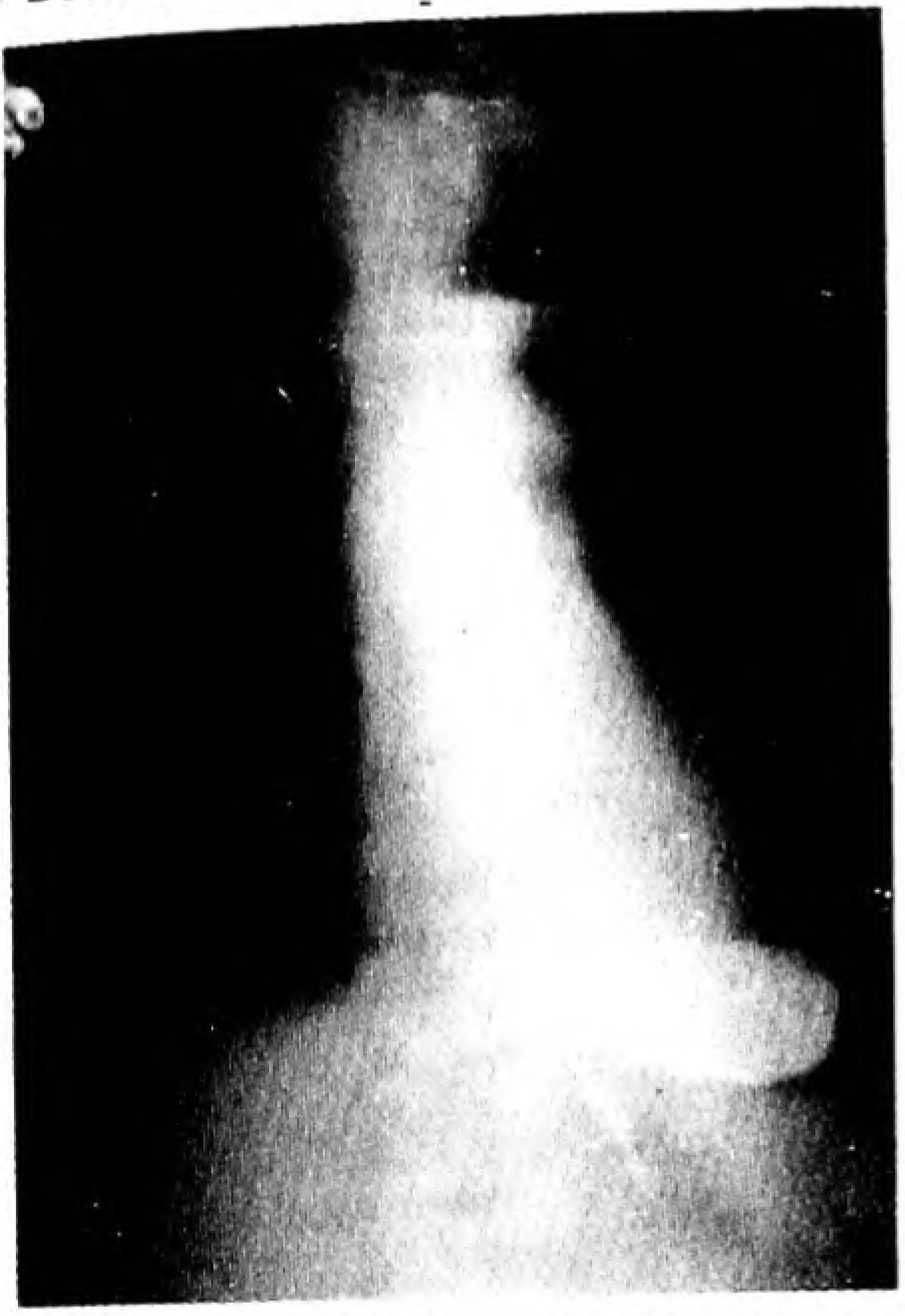

Fig.1: Single Contrast Barium Oesophagogram showing esophageal dilatation with an air fluid levels& smooth tapering at gastroesophageal junction. Gastric bubble is absent.

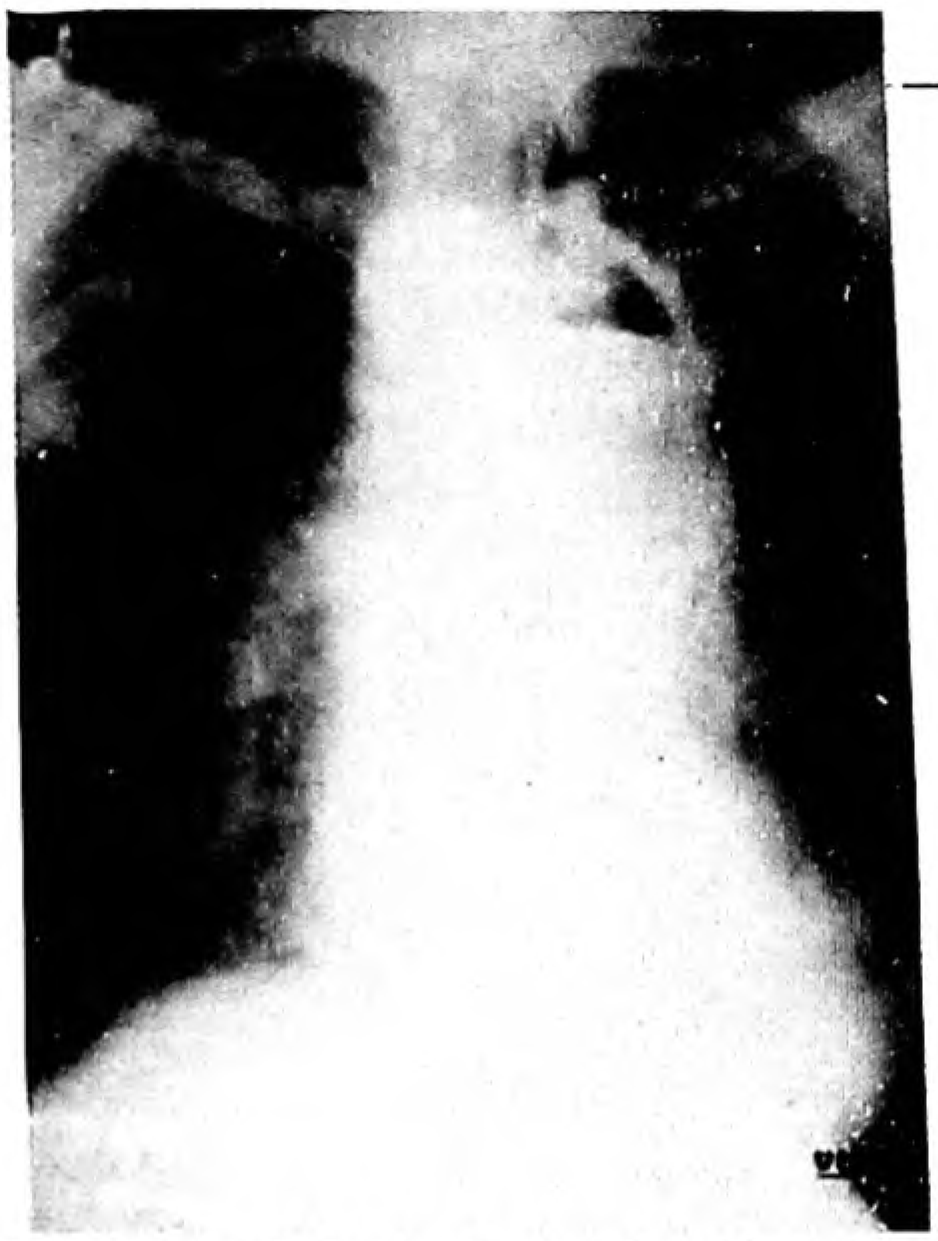

Fig.2: X-ray Chest (P.A. view) showing dilated oesophagus with air fluid levels.

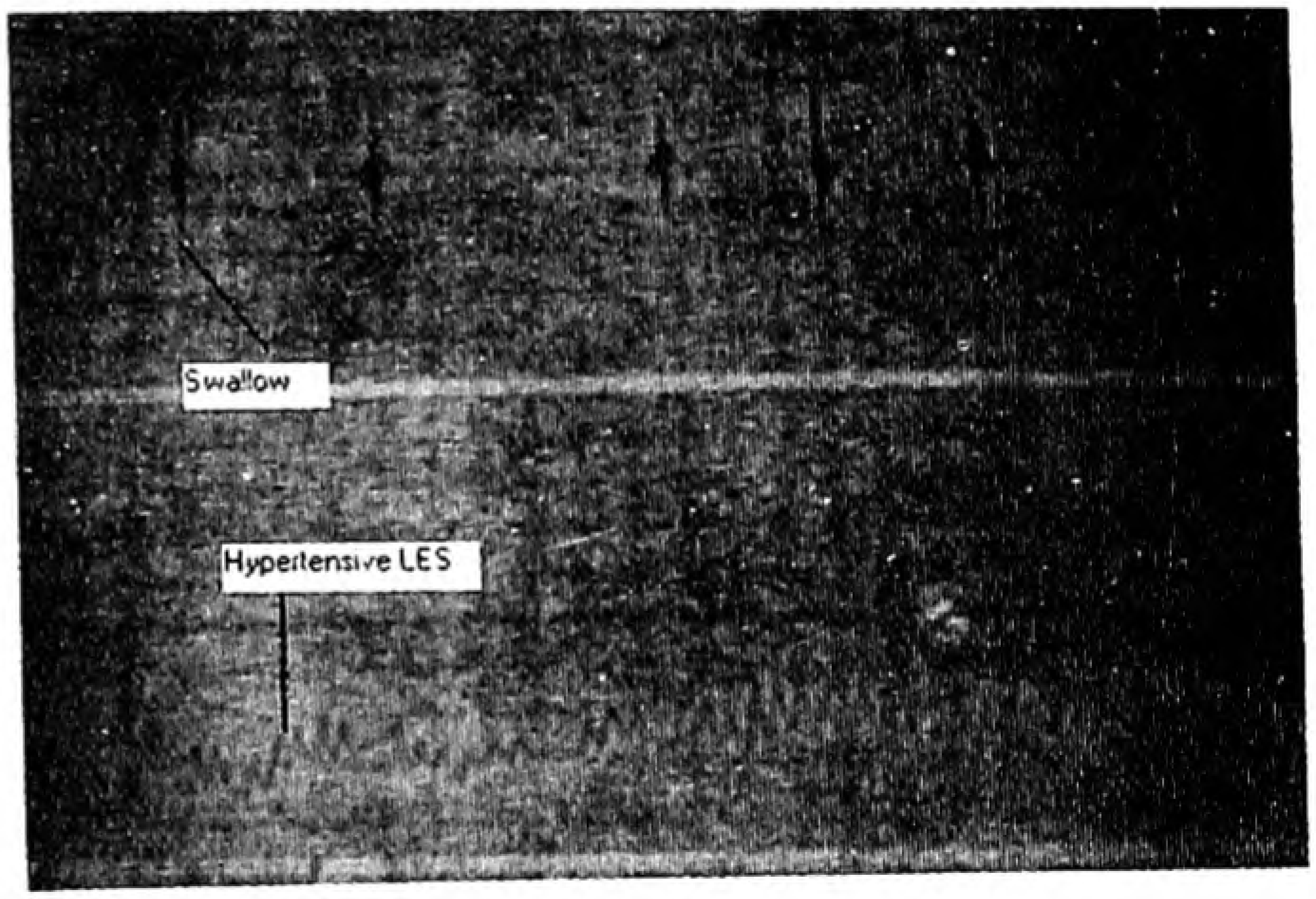

Fig.3: Esophageal manometry showing Hypertensive LES & failure of relaxation of LES to normal swallowing.

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PICTORIAL ESSAY
CURRENT STATUS OF ULTRASOUND IN BREAST IMAGING

Shiblee Hafeez; Masarat Syed; Arif Syed

Mammography is being considered the most common screening modality for the detection of breast cancer, but all mammographic abnormalities may not always turn out to be malignant.

Thus modern ultrasound has proved to be an ideal and accurate tool to complement mammography in the diagnosis of benign breast masses and thus avoid unnecessary and disfiguring surgical interventions for benign processes.

Breast US has proved helpful in benign breast problems and also with high resolution scanners and increased expertise of those operating the machines new and complicated problems are being handled confidently with breast US Most doctors now advocate Breast ultrasound as a more reliable tool than mammography as a follow up to a digital examination showing a palpable mass.

Any palpable mass can be accurately categorized as cystic or solid and to some extent a benign solid mass can be distinguished from a malignant one. Ultrasound is of particular value in:

1. Examining young breasts for which mammography is less informative.
2. Confirming the presence of a possible mass found by palpation or even seen on mammography.
3. Separating cystic from solid lesions.
4. To evaluate post surgical patients.
5. Diagnosing rupture in breast implants.

Ultrasound may also be used in conjuction with mammography as a follow up examination for patients who are at increased risk for breast cancer, such as those with a maternal, family history of breast cancer or a previous cancer in the other breast.

Sono mammography should be performed with high resolution linear array 7.5 to 10 MHz transducer. A high frequency transducer is desirable to obtain detailed view of the mass.

In order to perform good breast ultrasound, scanning experience is one of the most critical factors and it is really the only way to develop an appreciation of the strengths and the limitations of ultrasound in imaging the breast.

Specific Conditions

Cysts:

The most important use of breast ultrasound is its ability to diagnose a cyst with confidence. In the experienced hands the diagnosis of a simple benign cyst should be with close to 100% accuracy and thus to avoid unnecessary surgery or

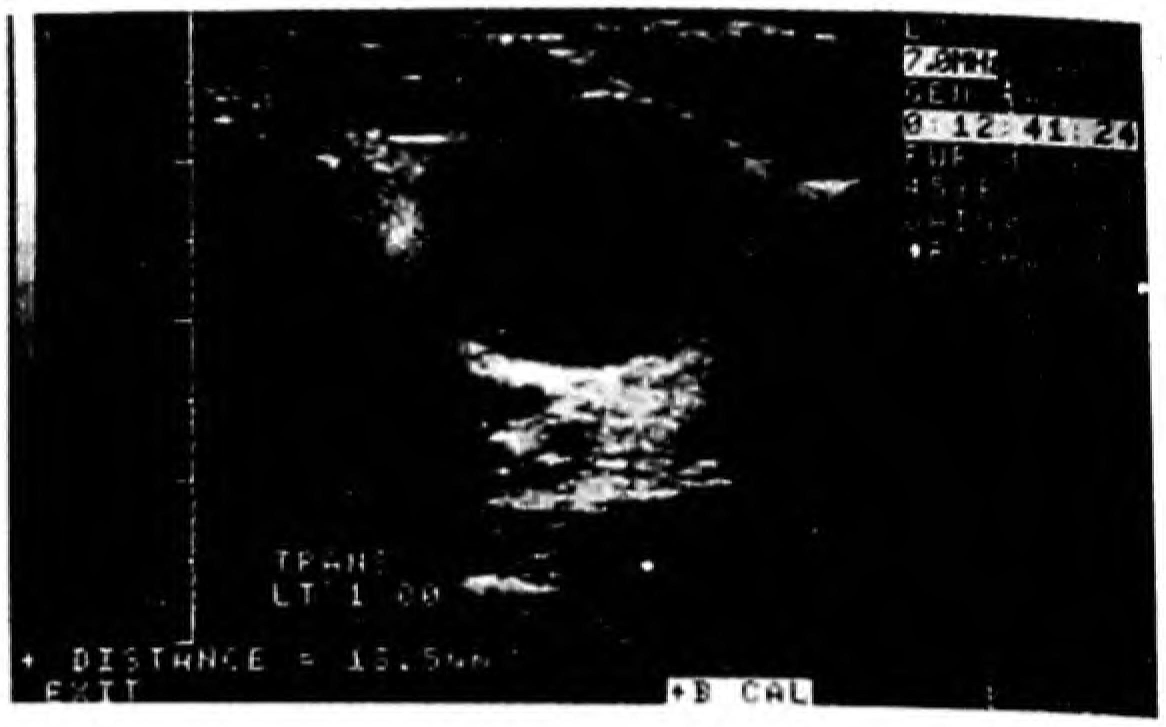

Fig. 1

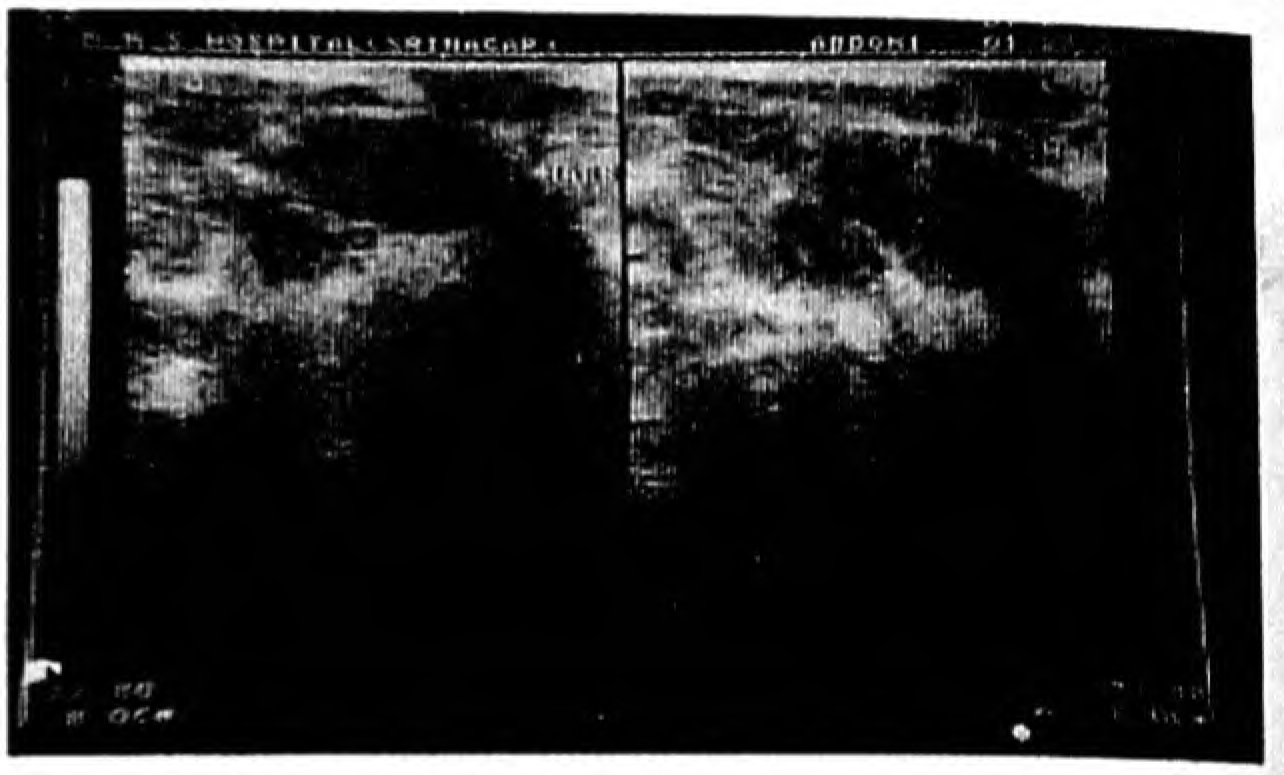

Fig. 2

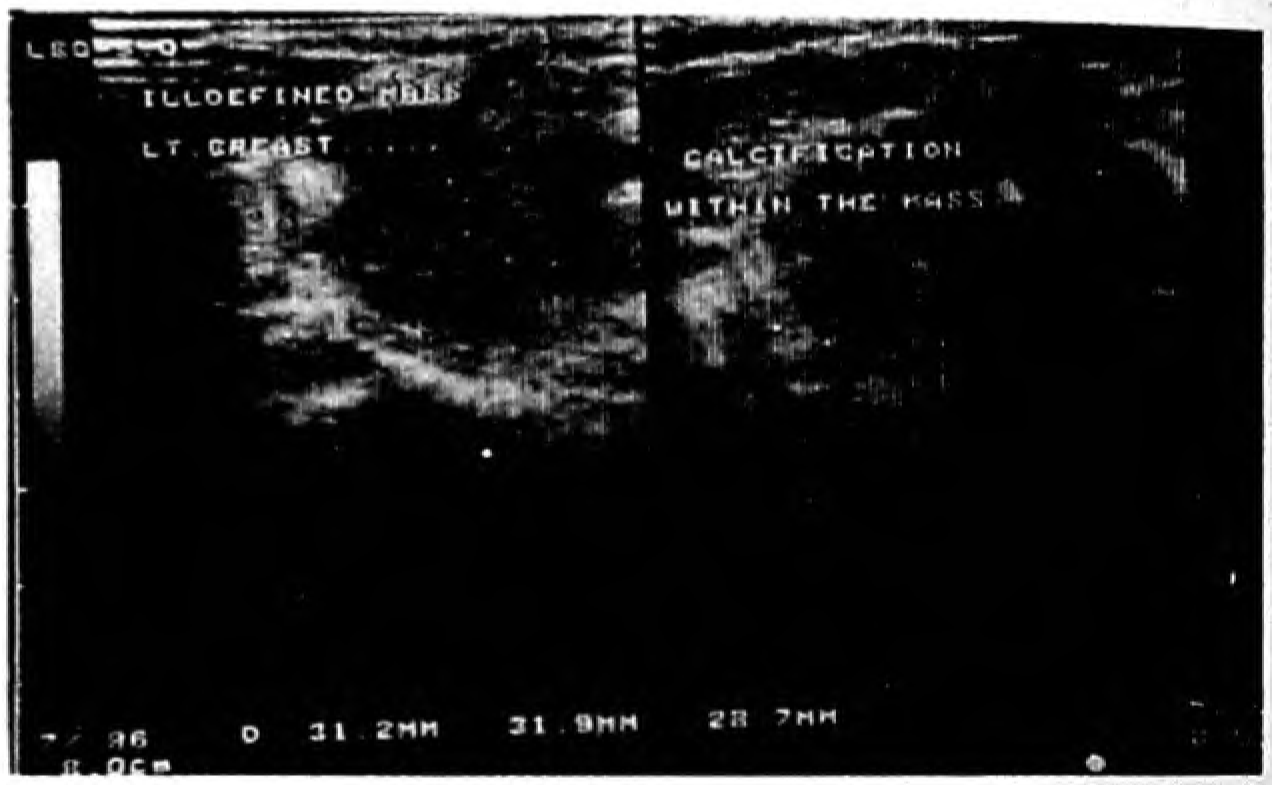

Fig. 3

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biopsy. A simple cyst usually has smooth walls, sharp borders, no internal echoes and distal acoustics enhancement (Fig. 1).

Sometimes the internal echoes may be produced by debris, haemorrahage infection or cholesterol crystals. A thickened cyst wall, mural nodules and papillary fronds are worrying features that may be due to cancer. Aspiration under US control is often done under such circumstances.

Solid Masses

Solid breast masses on USG may be divided into

Probably Benign Masses

Fibroadenomas are usually ovoid in shape with uniform low level internal echoes and a smooth rounded or mildly lobulated border (Fig. 2). Cystosarcoma phyllodes has a similar appearance but the mass is much larger and multiple cystic spaces within the masses can be seen. Benign features of a mass are:

Hyperechogenic
Ellipsoidal
Gentle Lobulations
Thin capsule
Compressibility

Probably a Malignant Mass

Cancers in the breast often have very obvious features that are very different from probable benign masses.

Cancers are usually hyperchoic and their shape usually is not smooth, but instead they have sharp multiple lobulations or even sharply angled borders. The lobulations may be micro or macrolobules. They do not have a predominant horizontal axis and may even be taller than wide (Fig. 3).

Cancers also demonstrate posterior shadowing and on rare occasions calcifications can be seen within the mass (Fig. 4).

Indeterminate Masses

Unfortunately about 25% of cases which were initially suspected to be benign on US turned out to be malignant. Thus ultrasound cannot confidently identify lesions as being malignant or benign in about 25% of cases. This increases the need for histopathological confirmation for any detected solid mass on USG.

Abscesses

Abscesses are fluid filled and usually contain some debris. They have well defined thick borders and tend to occur in the periaerolar area.

Galactoceles

A galactocele is a rare milk collection seen as a poorly outlined relatively echo free area with a few internal echoes.

Implant Problems

Breast reconstruction after mastectomy is done either with prosthesis or various musculocutaneous and fatty flaps.

Common prosthestic implants are those of silicone or saline filled bags. Mammographic evaluation is difficult and sonography and MRI are the modalities of choice to evaluate these implants. US can be used to evaluate some complications like rupture, displacement and capsule formation.

Sonographically Guided Procedures with needles

US can provide a quick reliable and accurate guidance for placement of needles into the breast for various types of interventional procedures. Cyst aspiration, needle localization of lesions for excisional biopsy and percutaneous core biopsy of the masses are being performed under ultrasound guidance with increasing frequency.

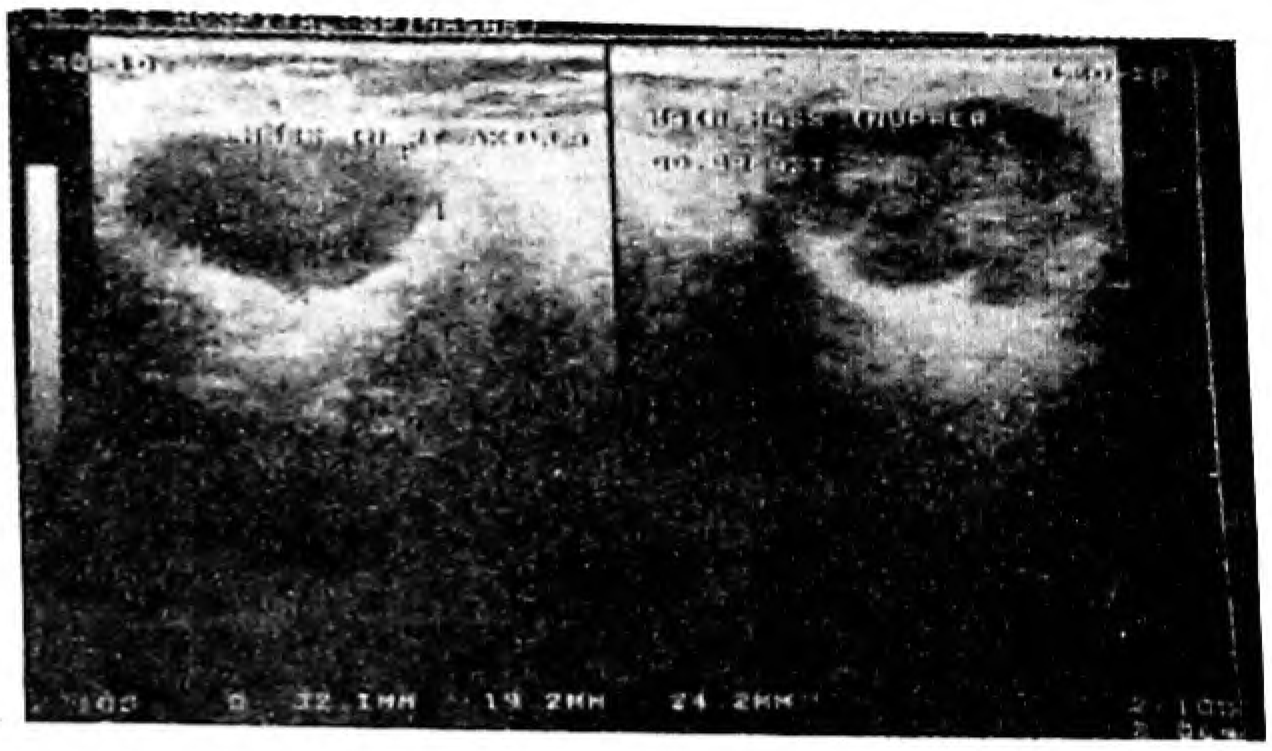

Fig. 4

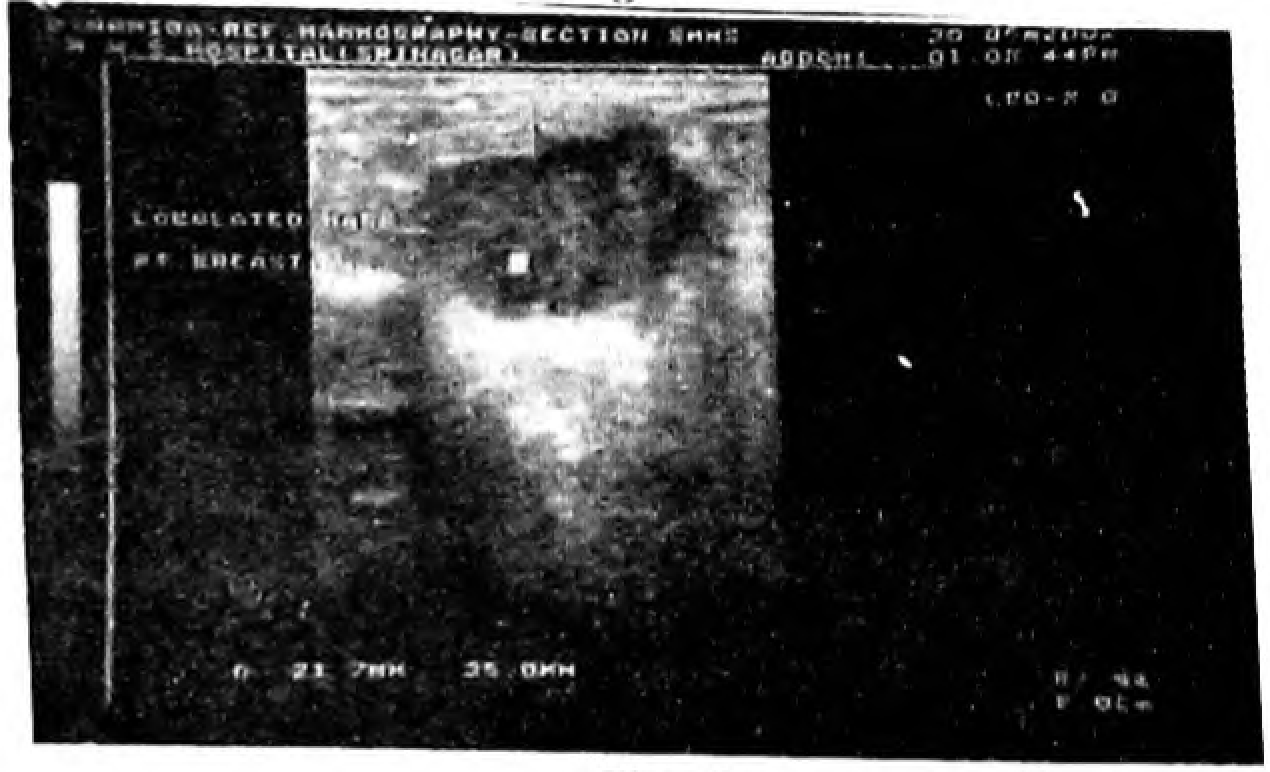

Fig. 5

Conclusion:

High resolution ultrasonography of the breast is currently the most useful modality to differentiate cystic from solid breast lesions. It is also more informative than simple film mammogaphy in imaging the young (glandular) and dense breasts. Also it has a definate role in distinguishing solid benign lesions from malignant breast lesions and thus avoids the unnecessary biopsies of benign lesions which add to the procedural risk, patient discomfort, anxiety and increased cost to be incurred by the patient.

References:

1. Wild JJ, Neal D. Use of high frequencey ultrasonic waves for detecting changes of texture in living tissue. Lancet 1951;1:655-657.
2. Marc J Homer. Mammographic interpretation, a practical approach. Breast ultrasound 2nd ed. 164-165.
3. Fornage BD, Lorigan JG, Andry E. Fibroadenoma of breast; Sonographic appearace Radiology 1989;172:671-675.
4. Marc J Homer. Mammographic interpretation, a practical approach. Breast ultrasound 2nd ed. 172.
5. Jackson VP Sonography of malignant breast disease. Semin Ultrasound CT MR 1989;10:119-131.
6. Tono E, Cosgrove DO, Sloane JP in ultrasound diagnosis of breast disease. Churchill Livingstone, 1994 pages 75-102 and 157-180.
7. Roger C Sanders Clinical Sonography. A practical Guide. Breast 3rd ed. 418.

Q & A WITH PROF. BASHIR AHMAD

Prof. (Dr) Bashir Ahmad, was born in the Apple Town of Shopian, After completing the Preliminary studies within the State, he did his M.B.B.S., from Maulana Azad Medical College and subsequently qualified for his Masters Degree in Ophthalmology from the same College in 1970. He worked as a Registrar for a brief period at Maula Azad Medical College and its attached Irwin & C.G. Pant Hospital. He joined State service as Registrar, Medical College, Srinagar, and gradually rose to the Post of Professor & HOD, Deptt. of Ophthalmology, Govt. Medical College Jammu & then Srinagar. He did his fellowship in the Anterior Segment of the Eye, from Institute of Ophthalmology and Moorfields Hospital London. He also did his fellowship from Institute of Clinical Ophthalmology Kirya Japan.

He is receipent of Man of the Millennium, Doctor of the Millennium, Best Ophthalmologist and Most popular teacher Award. He is presently the Director of Eye Care & Research Centre, which is among one of the best Centres in India. He has around 35 Publications in both national as well as international Journals.

Here are some of the questions which I asked to the Doctor, in an Interview.

Q.1. *Doctor I have gone through your centre & I am fascinated by its setup. It seems all the latest equipments, are available here, Please tell me why did you choose Ophthalmology as your future?*

Ans. I am in Ophthalmology just by chance. I was working as a House Physician, in the Deptt. of Medicine in Irwin Hospital, now named as L.N.J.P. Hospital, in Delhi. The HOD of Ophthalmology, borrowed me from Medicine Deptt as they were short of House Surgeons, in the Deptt. of Ophthalmology. He however refused to let me go back to Medicine Deptt.

Q.2. *How much has Opthalmology advanced in the last ten years?*

Ans. I don't think there is any field of Medicine, which has advanced so much as Ophthalmology, in last ten years.

Q.3. *Do you feel with advanced gadgets treatment for simple Surgery like Cataract or even a routine Eye-Checkup has become expensive, and is worth it?*

Ans. There is not doubt that the field of Ophthalmology has become extremely expensive both for the Ophthalmologist as well as for the Patients. The equipment is expensive & it needs a regular maintainance by a qualified engineers. But I feel the quality of Eye Care, has undergone a tremendous change and it has gone all the way in favour of the Patients.

Q.4. *For the last few years you have been in Private practice, Do you feel more Private Hospitals or Clinics, are needed at District level also?*

Ans. I personally feel that the health care system should be Privatised at every level, if one wants to give quality care, to the Patients.

Q.5. *Do you have any Suggestions for doctors, Practising in the Ophthalmology?*

Ans. I am sure the your doctors are very hard working. They should bear in mind that this is not Profession which is ment to be used for Commercial Purpose. They have to give their best to the Profession & be happy if they have restored the sight of a Visually handicapped person. They should remember that there is no money in this Profession.

Q.6. *As ex-president of Medical faculty, G.M.C. Sgr. what are your Suggestions to improve Medical Faculty & teaching in our Hospitals. Is medical ethics maintained as it was in the past or not?*

Ans. It is very unfortunate that in the last more than a decade, we have seen tremendous downslide in the health care facility as well as in teaching. We are capable of reaching to the top, we have best trained doctors. The only thing needed is hard work, dedication & sincerety. Although over whelming majority of the doctors are adhering by the medical ethics, but from last few years, unfortunately we have seen erosions taking place in this Vital Segment.

Q.7. *What is the scope of the private Medical Sector in our State, why still patients are runing away from State, for their treament to Delhi or Mumbai?*

Ans. There is tremendous Scope for Private Medical Sector, in our State. I am sure if all the modern facilities are provided here, there is no need for the Patients to go out side for the treatment.

Q.8. *Since your whole family is in medical profession, Do you think it was a right decision?*

Ans. I still feel this is the best way to serve the suffering humanity and I have no regrets about it.

Q.9. *Doctor you hail from a far flung village of Shopian and you have spnd your good portion of the time in G.M.C, Srinagar, what has been your contribution to your place of birth?*

Ans. I have done my best to provide the treatment of international quality to whole of the State, which also includes the town of my birth.

Q.10. *Last but not the least, is it worth to put lot of hard work and money in medical sector?*

Ans. As I already said that this is the Profession which requires give, give and give. If you have benefitted the patients the reward lies in their blessings, there is no financial benefit in this field.

Q.11. *What is your Opinion about JK-Practitioner?*

Ans. I have all my praises for the Journal. It has provided a medium of expression for all the docors belonging to different fields and every issue shows improvement over the previous one.

Q.12. *If you were given a chance in your next birth, what would you like to be?*

Ans. An Ophthalmologist.

Interviewed by ***Prof. G.M. Malik*** *Chief Editor JK-Practitioner*

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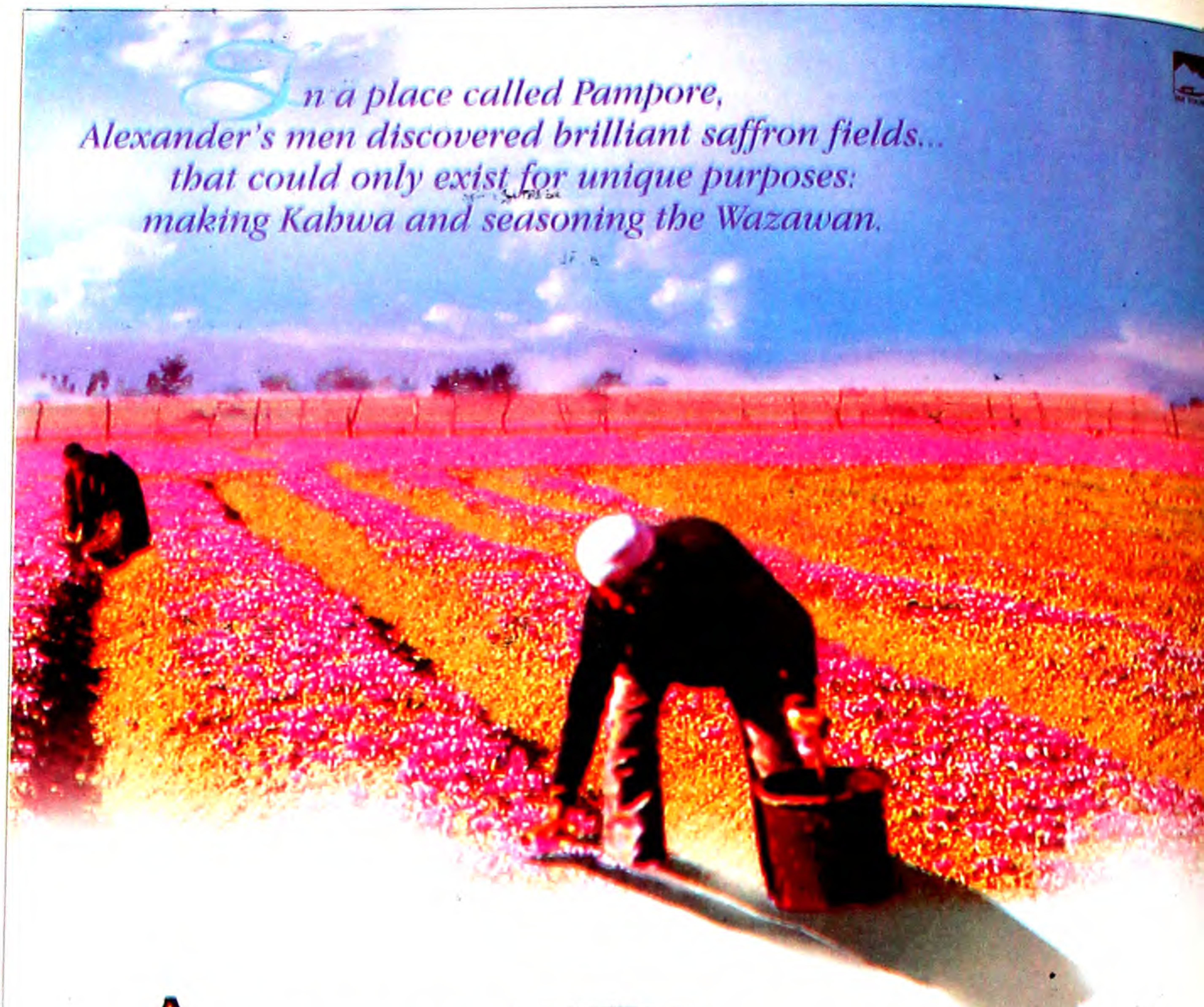
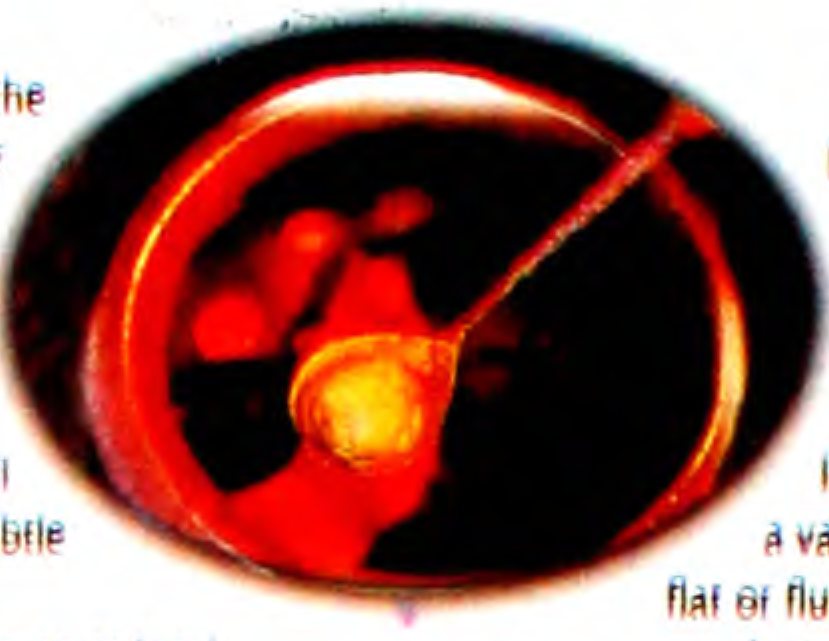


Ancient Persian literature recites the unforgettable joy and admiration of Alexander the Great's soldiers upon reaching Kashmir's sprawling saffron fields, after a 5000 km journey. The Greeks used the precious pink saffron in medicine, the Hindus for adornment. The unique Kashmiri tea – Kahwa – is incomplete without the subtle tint and flavour of saffron.



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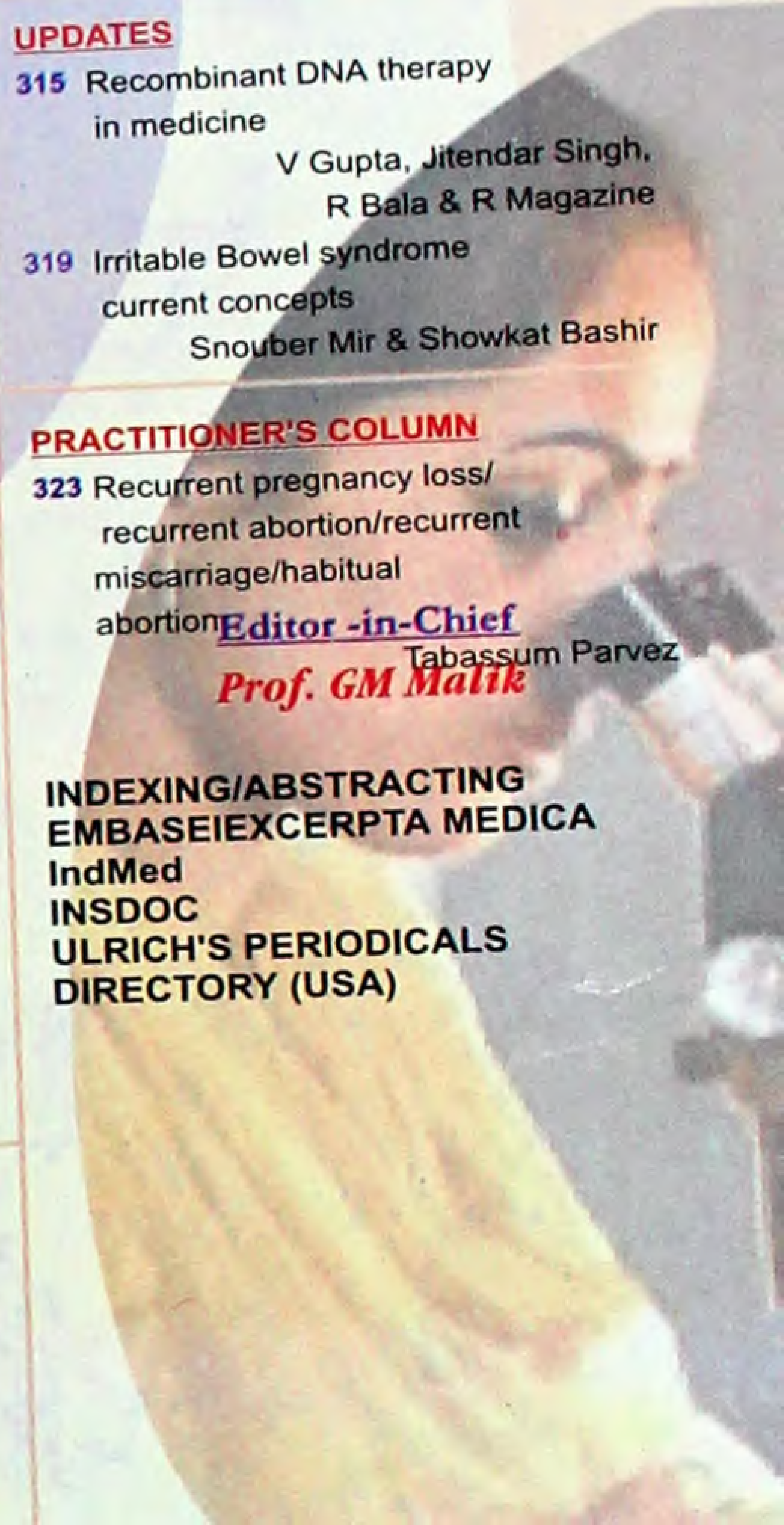

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CORTICO BASAL GANGLIONIC DEGENERATION (CBGD)

Ghulam Nabir Mir, B.Sc.. M.Pharm, MBA, Ph.D

Introduction

Corticobasal ganglionic degeneratioon (CBGD) is a rare progres-sive disease, meaning the symptoms worsen over time. Over the course to several years, most people with CBGD gradualy worsen, with symptoms beginning with and progressing to involve upper ad lower extremities. It is a neurological disorder charac-terized by a combination of Parkinson-like and cortical dysfunc-tion. CBGD is a rare, sporadic, progressive disorder first re-ported in 1968. CBGD appears to be closely related to another, less rare, progressive Supranuclear Palsy (PSP).

Several regions of the brain degenerate in CBGD. The cortex, or outer layer of the brain, is several affected, especially the fronto-parietal regions, located near the center-top of the head. Other, deeper brain regions are also affected, including parts of the basal ganglia, hence the name. In CBGD, cognitive symptoms dominate, while in PSP, eye movement symptoms dominate the pic-ture. Studies of brain tissue in individuals with CBGD shows certain characteristic cell changes. Similar, although not iden-tical, changes are observed in two other neuro-degenerative diseases: Pick's disease and Progressive Supranuclear Palsy, both of which involve a brain protein called tau. CBGD shows certain characteristic cell changes which have provided researchers some intial clues in their search for the causes.

CBGD is generally an asymmetric akinetic rigid syndrome, un-responsive to levodopa, similar to that of multiple system atrophy and PSP. Eye movement abnormalities are common, as in PSP, and supranuclear gaze palsy can be seen as in PSP. Given the genetic similarities between CBGD and PSP, it seeems possible that they are simply two "faces" of the same disease.

Neuroradiological imaging studies in CBGD demonstrate cortical atrophy, which may be symmetrical or asymmetrical. Other cortical signs include alien limb phenomenon, apraxia, dysphasia and cortical sensory loss Proposed diagnostic criteria include at least three of the following: bradykinesia and rigidity that does not respond to levodopa, alien limb phenomena, cortical sensory signs, focal limb dystonia, and action tremor myoclo-nus. The "alien limb" symptom is highly specific but it is not necessary for the diagnosis. Arm levitation resembling alien limb phenomena has been reported in PSP (Barclay et al. 1999), which certainly can also show focal limb dystonia and bradykinesia.

Causes of Syndrome

What causes the degeneration of brain tissue in CBGD? Unfor-tunately, the cause of CBD is entirely unknown. There is cur-rently no strong evidence to suggest CBGD is an inherited dis-ease, and no other risk factors, such as toxins of infections, have been identified.

Symptoms

What is the usual course of CBGD? A person with CBGD will usually become immobile due to rigidity within five years of symptom onset, and may require a gastrostomy tube for feeding at some point before that. Most often, within ten years of onset, pneumo-nia or other bacterial infections may lead to life threatening situations. CBGD usually begin after age 60. The initial symptos of CBGD are often stiffness, shakiness, jerkiness, slowness, and clumsiness, in either the upper or lower extremities. Other initial symptoms may include dysphasia (difficulty with speech generation), dysarthria (difficulty with articulation), difficul-ty controlling the muscles of the face and mouth, or walking and balance difficulties. Symptoms usually begin on one side of the body, and spread gradually to the other.

Summary of Symptoms

- Parkinsonism (rigidity, slow movements, postural instability)
- Tremor
- Myoclonus (sudden, brief jerky movements)
- Dystonia, including blepharospasm
- Speech difficulty

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- Mild-to-moderate cognitive impairment (memory loss, difficulty planning or executing unrehearsed movements, dementia)
- Sensory loss
- "Alien hand/limb" phenomenon (difficulty controlling the move-ments)

Other aspects of this picture could easily be mistaken for other neurodegenerative disease, such as Alzheimer's or Pick's disease, and, in fact, even experienced clinicians are correct only 50% of the time or less when judged by pathological criteria. Onset in the sixth or seventh decade is typical. Disease progression is quicker than in Parkinsonism, but similar to that of PSP. Recent-ly language disrturbance has been documented to be frequent (Frattali et al, 2000) Neuronal loss, gliosis, and swolen ach-romatic neurons (ballooned neurons) are found in all cortical layers, but especially in superior frontal and parietal gyri. There is extensive loss of myelinated axons in the white matter.

Scattered nuronal inclusions may be seen similar to Pick bodies.

Balloned neurons are strongly reactive for phosphorylated neurofilaments, and may include the tau protein (Dickinson et al, 1986). Neuronal loss and gliosis are also observed in the nuclei of the basal ganglia. Neurofibrillary tangles are absent. The substantia nigra shows neuronal loss with extraneuronal melanin, gliosis and neurofibrillary inclusions, called "corticobasal bodies."

Diagnosis:

CBGD is difficult to diagnose in its early stages, and experi-enced examiners typicallly diagnose it correctly less than 50% of the time (litvan et al, 1997) CBGD may also be impossible to differentiate from PSP or a striato-nigral type of MSA. As more cortical signs develop in later stages, the disorders may not be possible to separate. As diagnostic sensitivity is poor, neuro-pathological confirmation remains the goold standard. Even here, one wonders if this disorder can be defined or clearly differentiated from Parkinsonism, PSP (progressive supranuclear palsy, related by tau), MSA (multiple system atrophy) or Pick's disease.. While CBGD patients have normal saccadic velocity, this may be an artifact. If PSP and CBGD share the same pathologic mechanism, they may simply be two different presentations of the same disease.

The cause of CBGD is presently unknown but because the tau pro-tein accumulates in this disorder, it may be related to a muta-tion in the tau gene. (Higgins et al, 1999). Tau is a microtu-bule-binding protein that is normally abundant in neurons. Other "tauopathies" include Alzheimer's disease, Pick's disease, fron-totemporal dementia, Parkinsonism, and Progressive Supranuclear Palsy (PSP) (Higgins et al, 1999). According to Di Maria et al (2000) and Houlden et al (2001), CBGD shares the same tau haplo-type as do PSP patients, suggesting that both CBGD and PSP share the same genetic background and possibly the same pathologic mechanism.

Treatment

CBGD patients do not respond to levodopa treatmment (the standard treatment for Parkinsonism). Management is based on appropriate use of appliances, prevention of medical complications, and appropriate use of nursing. Patients with CBGD and caregivers should establish early the plan regarding invasive care - intuba-tion and feeding tubes, as these issues are almost certain to come up in the course of the disease. References: Physical therapy exercise may be useful to maintain range of motion of stiff joints This may prevent pain and contracture (muscle short-ening), and help maintain mobility. Occupational therapy may be used to design adaptive equipment that supports the activities of daily living, thus helping to maintain more functional independence. Speech therapy is usd to improve articulation and volume.

Authors Note:

I was diagnosed with CBGD on September 27, 2002 Although the symptoms started almost three years previously, changes were initially slow in progression. However, in the last months the syndrome has progressed rapidly. As I have been learning to deal with this condition, I was surprised to find that not much is known about the syndrome and not many neurologists are familiar with CBGD. In fact, the first neeurologist diagnosed my condition as MS.

Conclusion

Since September of 2002, I have been examined by numerous neurologists at three hospitals in Philadelphia: University of Pennsylvania, Jefferson Medical University, and Pennsylvania Hospital, The Cleveland Clinic in Ohio, and Stonybrook Medical School in New York. All diagnosed my condition as CBGD. On June 22, 2003, for two weeks I will participate in a clinical study of CBGD at the National Institute Neurological Diseases in Bethesda, Maryland.

References:

1. Barclay CL, Bergeron C, Lang AE. Arm levitation in progressive supranuclear palsy. Neuroloogyf 1999;52:879-882.
2. Di Maria et al. Corticobasal degeneraion shares a common genetic background with progressive supranuclear palsy. Ann Neurol 2000;47:374-377.
3. Dickson DW and others. Ballooned neurons in select neuroode-generative disease contain phosphorylated neurofilament epitopes. Acta Neuropathool 71:216-223, 1986) Neurology 2000;54:990-992.

4. Supranuclear palsy share a common halo type with corticobasal-basal degeneration. Higgins JJ, Litvan I, Nee LE, Lovel. Neu-rology 1999;52:404-406.
5. Houlden H and others. Corticobasal degeneration and progres-sive 2001;56:702-6.
6. Koller WC, Montgomery EB. Issues in the early diagnosis of Parkinson's disease. Neuroloogy 1997;49(Suppl 1), S10-25.
7. Litvan I, and others. Accuracy of the clinical diagnosis of corticobasal degeneration: a clinicopathologic study. Neurology 997;48:9-125.
8. Riley DE, Lange AE, Lewis A, et al. Cortico-basal ganglionic degeneration. Neurology 1990;40:12.
9. Kompoliti K, Goetz CG, Boeve BF, Maraganore DM, Ahlskog JE, Marsden CD, Bhatia KP, Greene PE, Przedborski S, Seal EC, Burns RS, Hauser RA, Gauger LL, Factor SA, Molhoo ES, Riley DE Cortico Basal Ganglionic Degeneration. Arch Neural 1998 Jul;55(7):957-61 Abstract.

HIRSUTISM - CLINICAL APPORACH

Mohd Ashraf Ganie M.D., D.M.

Hirsutism defined as the presence of male pattern tenainal hair in females affects 5-10 % of women[1]. The condition is extremely distressing and has ignificant negative impact on female psychosocial development[2]. In the last decade there has been a tremendous advancement in the management of hirsutism with renewed interest of specialists of internal medicine, from dermatologists, endocrinologists, gynecologists etc.

In the majority of patients, hirsutism should be considered as a sign of the underlying disorder like polycystic ovary syndrome (PCOS), androgen secreting lesions, non-classical congenital adrenal hyperplasia (NCAH), drug intake etc. After excluding these disorders the hirsutism is referred to as idiopathic, simple or peripheral. However this definition has evolved over the years and presently idiopathic hirsutism should be diagnosed in females with normal circulating androgen levels and normal ovulatory cycles, since 40% eumenorrhoic females have been demonstrated to have anovulatory cycles[3]. By this definition prevalence of idiopathic hirsutism is 5- 15%.

Clinical Evaluation of Hirsutism

This is the area of greatest discrepancy and controversy. An insufficient and insensitive evaluation will result in missed diagnosis including our opportunity to detect or minimize the impact of accompanying metabolic derangements like Type 2 Diabetes mellitus. Alternately obtaining excessive number of tests would lead to con and healthcare burden (e.g. 105, 905, 100 $ in USA are spent annually on 7% Of reproductive age group women who are hirsute).

Thus, for establishing an accurate diagnosis, a clear history and apt ' I examination are cardinal in the evaluation. Most important is to confirm that the patient is actually hirsute. Although unwanted facial or body hair can be distressing, it should be attended to only if excess terminal hair is in male-like pattern signaling androgen excess.

Clinical diagnosis is relatively subjective, however, one should determine weather hair growth is terminal or vellus and weather it is male in pattern. Feriman and Gallywey originally described the visual method of assessment by scoring 11 different body areas (chin, upper lip, chest, upper abdomen, lower abdomen, arm, forearm, thigh and lower leg). This was later on modified by excluding arm and leg areas[4]. In each of these areas hair growth is scored from 0 (absent terminal hair) through stage 4 (extensive terminal hair). Scoring of hair growth in sideburn area, lower jaw and upper neck and buttocks have been included in more recent scoring systems[5].

Ferriman Gallwey score in 161 women showed that 9.9% had a score >5, 4.3% had score >7 and 1.2% had score > 10. Thus they reccmmended a score of >8 should be considered hirsutism.

Diagnostic Evaluation

For diagnosis of PCOS, the commonest cause of hirsutism, Na ' Institute of Child Health and Human Development (NICHD)[6], published following criteria:-

a. Clinical and/or biochemical hyperandrogenism
b. oligo-ovulation
c. exclusion of disorders like hyperprolactinemia, NCAH, Cushing's' syndrome and thyroid dysfunction. Demonstration of oligo-ovulation is important as 40% of hirsute women with regular menstrual cycles have ovulatory dysfunction[3]. Hypothyroidism and hyperprolactinemia should be ruled out, as both , conditions are known to cause an ovulation. TSH estimation is mandatory as hypothyroidism can be a cause of hypertrichosis (vellus hair growth) also. Cushing's syndrome and androgenic drug use also should be ruled out as hair growth can occur in both settings.

Serum gonadotrophin levels generally are unnecessary for diagnosis of PCOS because only 50-60% patients have elevated ratio (i.e. LH/FSH > 2) and normal LH/FSH ratio can't exclude PCOS. Androgenemia demonstration in a grossly hirsute patient is also unwwry. Normal androgen levels don't exclude PCOS because of variation in assay techniques and/or normal ranges in various laboratories. However free testosterone has a definite role in a minimally hirsute or non-hirsute oligo-ovulatory patient. Serum testosterone level > 200 ng/ml and DHEAS > 6000 ng/ml suggests androgen secreting tumor of ovarian or adrenal origin[7] which in renal adenoma or carcinoma, ovarian

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arrhenoblastoma or Krukenbergs tumor. For excluding NCAH due to 21-hydroxylase deficiency (1-8% incidence) 17-hydroxyprogesterone in preovulatory period should be done and if it is > 2 ng/ml, an ACTH stimulation test should be done. This not only is important for diagnosing PCOS but also is relevant to give preconception counseling to patients. NCAH due to 3p-HSD and 17p – hydroxyls deficiency needs no investigation, as they are practically nonexistent.

All hirsute females should be screened for diabetes, as 30% of PCOS have impaired GTT and 8% have frank diabetes mellitus. Fasting and GTT suppressed insulin levels should be done to indicate any evidence of hyperinsulinemia and insulin resistance, which on one had, indicates the cause of hyperandrogenemia and on the other hand shows potential benefit of insulin sensitizers to prevent the future risks of the syndrome. Although screening for diabetes mellitus is cost effective, the benefit of improving insulin sensitivity parameters is not yet clear.

Imaging techniques such as USG pelvis and adrenals although thought to be of pivotal importance in diagnosis of PCOS, may not be needed routinely. Not all females with cystic ovaries have PCOS or vice versa. Transvaginal USG is misleading although adnexae may be easily seen.

Management

Hirsutism of sufficient severity should be investigated promptly for reasons of negative impact on psychosocial behavior and for excluding any underlying life threatening disorder especially in the presence of any additional features of defeminization or virilization. After ruling out any androgen-secreting lesion, management plan can be made on symptomatic basis. Although hirsutism is the prime concern of patients, syndromic accompaniments like acne vulgaris, oligo-ovulation, and insulin resistance should be targeted. This approach can be summarized as follows.

Cosmetic therapy

Cosmetic therapy is directed to remove or conceal the hair in exposed areas of the body. Depilatory (surface removal) techniques like shaving or chemicals can be used. Shaving, although has no effect on coarseness or longitudinal growth of hair it is unacceptable to many ladies due to coarse feel of blunt ends. Most chemical depilators, which are substituted mercaptans, reduce disulfide bonds of peptide chains in keratin, causing softening of the hair, which later can be washed off. These methods, if employed properly are safe.

Epilatory methods like plucking and wax therapy are temporary and need multiple sittings in addition to being painful. Permanent epilation can be done by electrolysis (40% hair recurrence) short wave diathermy or laser. Laser therapy uses 694 °A non Q switched rays which cause photo acoustic shock wave damage to hair follicle in melanin secreting cells (sparing nonpigmented telogen bulbs). Ruby laser is most suited although response is less in dark skinned people. Approximately 3 sittings of treatment are needed to have all hair in anagen phase. Although mild scabbing and discomfort can occur, scarring is rare. The success of therapy is operator dependent and in a 2-8 weeks study 64% females described it as satisfactory or very satisfactory[8].

Pharmacotherapy

Antiandrogens, which act at many places in the androgen pathway, are commonly used in the treatment of hirsutism. Most drugs take 6 to 9 months before any effect is detectable and the effect lasts only till the drugs are given i.e. they are suppressive but not curative. Oral contraceptive agents (OCP's) decrease ovarian androgen production via suppression of hypo-thalam-opituitary axis and also help to regulate menstrual irregularity induced by antiandrogens. The OCP's with cyproterone acetate has been used with good success (Diane 35). These have also been used in combination with GnRH agonists (e.g. buserelin), flutamide or finasteride with better efficacy than antiandrogens alone[9]. Spironolactone, a synthetic steroid related to aldosterone acts by ~king DHT receptors competitively. Response is good and occurs , n 2 – 12 months of use, causes reduction in anagen hair and Ferriman Gallwey score significantly. The usual doses employed are 50 – 300 mg/day[10]. The side effects are menstrual irregularity, postmenopausal bleed, breast tenderness, hyperkalemia, fatigue and emasculation of a male fetus. Cyproterone acetate, an androgen receptor blocker has potent progestogenic and antigonadotrophic effects. The efficacy is comparable to spironolactone although weight gain, nausea, loss of libido, breast tenderness and emasculation of male fetus are side effects. The usual recommended dose is 50 mg/day 10 days a month, although some authors advocate a starting dose of 100 mg/day and to be co-ordinated with menstrual cycle[11].

Flutamide, a non-steroidal agent that acts by inhibition of androgen uptake and nuclear binding has been used for treatment of prostatic carcinoma. It is now recommended for hirsutism as many studies have demonstrated a significant benefit with 250 – 500 mg/day dosage. One study has demonstrated efficacy superior to spironolactone[12]. Minor side effects like dry skin, hot flush's, deereased libido, fatigue, gastric disturbances, menstrual irregularities do occur but fortunately dreaded side effect like hepatotoxicity is rare (<1%) and thus mandates periodic liver function testing.

GnRH agonists like leuprolide acetate and buserelin are used to saturate pituitary receptors continuously leading to decrease in estrogen and androgen levels via LH and FSH fall. These drugs are given parentally and have to be combined with OCPs in view of hypoestrogenism. Side effects are loss of libido, migraine like headache, emotional liability and decrease in bone mineral density. These drugs are to be used in severe &. non- responsive cases only.

Finasteride blocks 5 u-reductase (although primarily type 1 isoenzyme of genital skin and prostate), and has established benefit in hirsutism after 6 months of therapy[13]. Side effects known are headache, breast tenderness, depression and hypersensitivity. Thus it appears safe and promising although further study is needed.

Insulin sensitizers currently introduced to correct hyperinsulinemia/insulin resistance, the underlying, and mechanism of hyperandrogenism in PCOS. Troglitazone and metformin have been shown to have promising results in reversing the all components of the syndrome. Metformin has been recently shown to reduce androgenism and resume ovulatory cycles in 60 – 80% of patients[14].

In conclusion hirsutism is a common disorder and needs systematic approach to diagnosis and therapy. Many investigations currently done may be Unnecessary and therapy should be attempted to correct the underlying disease process rather than problem oriented therapy used traditionally.

References:

1. McKnight E The prevalence of "hirsutism" in young women. Lancet 1964 1:410-413
2. Barth 3H, Catalan 3, Cherry CA, Day A 1993 Psychosom Res 37:615-619
3. Mehta et al f1992f: Should androgenlevels be measured in hirsute women with normal menstrual cycles? Int.3 Fertil 37: 354-357.
4. Ferriman D, Gallwey 3 D 1961 Clinical assessment of body hair growth in women .3 Clin Endocrinol Metab 21:1440-1447.
5. Redmond GP 1995 Clinical evaluation of the woman with an androgenic disorder. In: Redmond GP (ed) Androgenic Disorders. Raven Press, New York, pp 1-20.
6. Zawadzki 3K, Dunaif A 1992 Diagnostic criteria for polycystic ovary syndrome: towards a rational approach. In.Dunaif A, Givens 3R,Haseltine F, Merriam GR (eds) Polycystic Ovary Syndrome. Blackwell Scientific Publications,Boston,MA,pp 377-384.
7. Ricardo Azziz (2000): The time has come to simply the evaluation of hirsute patient. Fertil Steril 74: 870-872.
8. Zachariae H, Bjerring P, Lykakee H (1997): Laser epilation. A preliminary. investigation Aust 3 Dermat. 38: (Suppl 2);S 52.
9. Carr BR, Breslau NA, Givens, C (1995): Oral contraceptive pills, GnRH agonists are use in combination for therapy of hirsutism. A clinical research center study. 3CEM 80:1169-78.
10. Burke BM, Cunliffe W3 (1985): Oral spironolactone therapy for fernale with hirsutism or androgenic alopocia. Br 3 Dermatol 112:124-125.
11. Hammerstein 3,Meckis 3, Leo Rossberg I(1975): Use of cyproterone acetate in the treatment of acne,hirsutisrn and virilism. 3 Ster.8iochem Mol Biol. 6: 820-836.
12. Fruzzetti F, De Lorenzo D, Ricci C et al 1993.Clinical and endocrine effects of flutamide in hyper endrogenic women.Fertil Steril 60:806-13
13. Ciotta L, Cianei A, Calojein et al (1995): Clinical and endocrine effects of Finasteride in women with hirsutism. Fertil Steril 64:299-306.
14. Paolo Moghetti, Robeto C, Carlo N (2000): Metformin effects on clinical features, endocrine and metabolic profile and insulin sensitivity in PCOS. A randomized, double blind placebo controlled six months trial followed by open long-term clinical evaluation. 3CEM 85:139-146.

ESOPHAGOGASTRIC CANCER - THE CHANGING PATTERNS

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Esophagogastric Cancer:

The esophagogastric region includes tumorus of three distinct areas:(1)

1. Esophagus
2. Esophagogastric junction
3. Stomach

Esophageal cancer:

Esophageal cancer ranks fifth as a cause of cancer mortality: most cases occur in the developing countries.(1) Cancer of the esophagus is one of the commonest causes of death in adults in North-east Iran, where the incidence of this cancer is among the highest in the world.(2) Two types of cancers occur in esophagus, squamous cell carcinoma which develops from the squamous lining and adeno-carcinoma arising from the meta-plastic columnar lining known as "Barrett's Oesophagus".(1)

In Western world there is a steady increase in the adeno-carcinoma of esophagus since 1980's. In earlier studies by Turnbull(3) who reviewed forty-years of experience with carcinoma of esophagus at the Sloan-Kettering cancer centre an incidence of 24% was found for the adenocarcinoma of esophagus with a male:female ratio of 3:1 as compared with 4:1 in patients with squamous cell carcinoma. Of 45 patients with primary adeno-carcinoma of esophagus 2 were in the cervical, 8 in the upper thoracic, 21 in the mid-thoracic(14) and in the distal thoracic. Smithers(4) in his study found the frequency of adeno-carcinoma to 7.3% whereas for Bosch et al(5) it was 3.7% and for Webb and Busutil(6) it was 1.7%. In contrast in a review by Hesketh(7) of the tumour registry files from 1980 to 1986 at two Boston University Hospitals showed that 31% of the esophageal cancers were adeno-carcinomas comprising 81 cases while squamous cell cancer comprised 165 (63%) cases out of a total of 262 cases. In addition, data obtained from the tumour registries of the Commonwealth of Massachusetts and the State of Connecticut and the National Cancer Institute's SEER program also strongly suggest that esophageal adeno-carcinoma is increasing in frequency. The sub-site involvement for oesophagus in Hesketh's study was:

U (Upper)	0
M (Middle)	5
L (Lower)	68
US (Unknown or unspecified)	6
Total	79

**Two tumours were removed from the study.*

This study showed that the lower oesophagus was the most frequent site for the occurrence of adeno-carcinoma. Lower esophagus was further divided into true lower, gastro-oesophageal junction (extension into proximal stomach but with <50% gastric involvement) and gastric (>50% tumour volume involving the stomach). From middle esophagus only 2 tumours involved the true lower oesophagus whereas from the lower oesophagus 38 involved the true lower, 22 involved the GE junction and 8 involved the stomach.

Johnston BJ(8) in 1991 in a retrospective review on endoscopic records for cancer of esophagus from 1976-1990 reported 49% of all oesophageal cancers to be adeno-carcinomas. In an extended study from 1991-1995 adeno-carcinoma of esophagus represented an incidence of 70% which was the highest incidence reported till that date.

Devesa(9) in 1998 in a population based incidence data from National Institute's SEER programme calculated age adjusted incidence rates for carcinoma oesophagus from 7, 3 year time periods beginning in 1974-1976 and extending it through 1991-1994. It was found that the incidence of esophageal adeno-carcinoma surpassed that of squamous cell carcinoma. The annual rates per 100,000 population for adeno-carcinoma rose from 0.7 during 1974-1976 to 3.2 during 1991-1994, an increase of 350%, the corresponding numbers of cases rose from 164 to 934. During the same period the rates for squamous cell carcinoma of esophagus declined from 3.4 to 2.2. Among

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black males, increases were apparent for esophageal adenocarcinoma with rates increasing from 0.4 to 0.6. The rates for squamous cell carcinoma among black males continued to decrease into the 1990'2. Devesa concluded that increase in cigarette smoking might contribute to the trend through an early state. Carcinogenic effect alongwith obesity which might predispose to gastro-oesophageal reflux. In short, the incidence of adeno-carcinoma of esophagus has been increasing over the last three decades and probably reflects a genuine increase in the incidence of its recognized precursor lesion, Barrett's metaplasia.(10)

Oesophagogastric Junction and Cardiac Caners

Adenocarcinoma arising at the oesophagogastric junction pose many problems. They are difficult to classify as they can arise from the columnar lined lower oesophagus, from the cardia itself or from the gastric body/fundus, with an upward spread to involve the esophagus.(11)

Because of similar trends in the incidence of adenocarcinoma of the esophagus and cardia which have been found to be similar in their changing incidence, age at diagnosis, male:female ratio etc; adenocarcinomas of lower end of esophagus have been clubbed together with carcinomas of cardia and it is believed that it may be a similar disease(12) because while the incidence of gastric cancers is decreasing world-wide, the cancers of cardia are on an increase following the trend of the oesophageal adenocarcinoma.

In an autopsy study at the University of Dusseldorf(13) from 1950-1982, cancer of distal oesophagus accounted on an average for about 40% of all esophageal cancers whereas the percentage of cancers of the cardia among all gastric cancers was 15.4% on average. While the incidence of gastric cancer was found to decrease overall, a relative increase in cancers of cardia was found.

It has been suggested that adenocarcinomas of the lower esophagus and gastric cardia should be reclassified as esophagogastric junction (OGJ) cancers.(14) There are distinct differences in the sex ratio, age at diagnosis, and distribution of histological types for tumours arising within the esophagus, the oesophagogastric junction (OGJ) and the stomach. These features support the concept that disease arising at the OGJ and including the lower esophagus and/or gastric cardia should be considered an entity separate from disease that arises from within the esophagus and the stomach.

Cancer registry and hospital data shows that there has been a change in the topography of gastric cancer and topography and morphology of oesophageal cancer with an increasing proportion of cases of adenocarcinoma of the gastric cardia and lower oesophagus i.e. adenocarcinoma of oesophago-gastric junction.(7,15,16) A consensus conference of the International Gastric Cancer Congress declared that cancers arising at or close to the oesophago-gastric junction should be considered separately from gastric and other oesophageal cancers and a new classification system for junctional cancers was devised (Table-1).

Table-1: Classification of junctional cancer types (from Siewert and Stein, 1998).

Junctional Cancer	Description
Type-I	Adenocarcinoma of the distal oesophagus arising in an area of specialised intestinal metaplasia and which may infiltrate the oesophago-gastric junction.
Type-II	True carcinoma of the cardia arising from the cardiac epithelium or short segments with intestinal metaplasia at the oesophago-gastric junction, this entity is also often referred to as "junctional carcinoma".
Type-III	Sub-cardial gastric carcinoma while infiltrates the oesophago-gastric junction and distal oesophagus from below.

The death rates from cancers off the esophagus and gastro-oesophageal junction, adjusted for age have risen steadily since the early 1970's (from 3 to 6 per 100,000 and from 1.5 to 3 per 100,000 population in the United Kingdom respectively).[17]

Esophageal/gastro-oesophageal junction cancer and Barrett's metaplasia:

In Barrett's esophagus, the distal oesophagus is covered with a metaplastic columnar epithelium that predisposes to the development off adenocarcinoma. Initially, a segment of at least 3cms was required but the definition now also includes short segments(18) and finger-like extensions of metaplasia from cardia into the oesophagus. However, the increased risk of cancer correlates with the length of the metaplastic segment and the presence of specialized intestinal metaplasia with Alcan-blue positive goblet cells.(19) The incidence of intestinal metaplasia of both the oesophagus and the gastro-oesophageal junction are also increasing. This metaplastic tissue besides having a pre-malignant potential is related to bile and acid reflux disease.(10) About 8% of patients undergoing routine endoscopy and 3% of the adult population have at least 1cm of Barrett's esophagus.(20) Furthermore, 17% of patients undergoing routine endoscopy and 6% of the adult population may have intestinal metaplasia of the gastro-oesophageal junction(20,21) Metaplastic changes may progress from dysplasia to adenocarcinoma.(18) About 5-15% of people with Barrett's esophagus and 2-5% of those with intestinal metaplasia of the gastro-oesophageal junction also have dysplasia, which in case of Barrett's oesophagus increases the risk of cancer between 30-fold and 150-fold,(17) though the risk of cancer for people with metaplasia of the gastro-oesophageal junction has so far not been quantified.

The specialized epithelium may play a role in carcinogenesis at the cardia, but due to the high prevalence of intestinal metaplasia, it does not help in predicting a high risk of cancer at this level.(1)

Rare inherited syndromes of familial gastro-oesophageal cancer can occur though these are rare and account for only 1-5% of cases.(22)

Inherited germline mutations of the O.Ecadherin gene, involved in cell adhesion, leads to loss of O. Ecadherin expression which is not only a cell adhesion molecule but also a tumour suppressor gene. Reduced expression of adhesion molecules on the surface membranes of cancer cells make them far more likely to have invasive properties. This situation seems to occur during the progression from metaplasia to adenocarcinoma.(23)

Various other aetiological factors for metaplasia adenocarcinoma sequence have been suggested which include smoking, generalized use of anti-secretory agents, gastritis and helicobacter pylori infection.(1)

Carcinoma Stomach:

Gastric carcinoma is a world-wide disease. Its incidence, however, varies widely, being particularly high in countries such as Japan, Chile, China, Portugal and Russia and is four-fold to six-fold less common in United States, United Kingdom, Canada and Australia.(24) In most countries, there has been a steady decline in both the incidence and mortality of gastric cancer over the past six decades. In 1930, gastric cancer was the most common cause of cancer death in the United States, since that time the annual mortality rate in the United States has dropped from about 38 to 7 per 100,000 population for men and from 28 to 4 per 100,000 for women.

Although there has been a general decrease in the incidence of gastric cancer which correlates with a decrease in the antral cancers, an increase in the carcinomas of cardia has been noticed.(25) As has been pointed out earlier cancer of cardia is different in the sex ratio, histological type, prognosis and in relation to aetiological factors such as the consumption of alcohol and tobacco, when compared with gastric cancer in other locations. These differences have led some investigators to consider cancer of cardia as a separate entity.

Many studies were conducted, to determine changes in various aspects of gastric carcinoma comparing specified 3, 4 or 5 year intervals. In one such study by Craanen (1992).(26) comparing two time periods a significant increase in the proportion off patients with cardiac carcinoma was found in the second group (patients from 1973-1980 were assigned Group-I and these from 1981-1988 as Group-II), which correlated with an increase in the proportion of patients with diffuse carcinoma with a corresponding decrease in the intestinal type.

To explore the factors that have influenced a decrease in the incidence rate of gastric carcinoma, a study in Japan by Inoue.(27) was performed to note the changing pattern in clinico-pathological features of gastric carcinoma. Proportional distribution was compared over the three different periods (1965-1973, 1974-1982, 1983-199) according to the clinico-pathological features of the gastric carcinoma i.e. sex, age, lesional location, histopathological type etc. A decline in the carcinoma of antrum with a relative decline in the well differentiated adenocarcinoma and an increase in cardiac carcinoma was found. This changing pattern of gastric carcinoma in Japan was probably related to exposure patterns of same epidemiological factors e.g. Westernizing diet. The incidence of gastric carcinoma has thus declined, the factors which have contributed to this decline are as underlined in Table-II.

Table-II: Factors contributing to a decrease in the incidence off gastric cancer.

1. Decrease in intake of salty foods, especially slated fish, dried fish and pickled vegetables.
2. Reduction in the over-intake off carbohydrates such as rice.
3. Increase in intake of fresh and green-yellow vegetables.
4. Increase in protein and fat intake.(28)

The other factors contributing to gastric cancer like environmental, hereditary factors and association with H.pylori have not shown a change in the already existing pattern. However, in some studies Castellanos (1992)(25) while studying the changing pattern of gastric cancer in Oxfordshire over two time periods (1960-1964 and 1984-1988) found chronic active gastritis associated more with tumours of Lauren's intestinal type than with diffuse type (in 1980's) a similar trend was found in 1960's also, in the 1960-1964 series. In the same study a strong association was noted between the antral carcinomas and H.pylori infection. H. pylori infection was present in 80% of antral tumours in 1960's and in about 40% in 1980's which may relate to rising standards of living and improved public health.

REFERENCES

1. Lambert R. The role of endoscopy in the prevention of esophago-gastric cancer. Endoscopy 1999. 31(2): 180-199.
2. Castle V. Oesophageal lesions in Northern Iran: A premalignant condition? The Lancet 1979; 4: 217-220.
3. Turnbull ADM. Primary adenocarcinoma of the oesophagus Cancer 1968; 2: 915-918.
4. Smithers DW. Adenocarcinoma of the oesophagus. Thorax 1956; 11: 257.
5. Bosch A, Fias Z. Adenocarcinoma of the oesophagus. Cancer 1979; 43: 1557-1561.

6. Webb JN, Busuttil A. Adenocarcinoma of the oesophagus and of oesophagogastric junction. Br J Surg 1978; 65: 475-479.
7. Hesketh PJ, Clapp RW. The increasing frequency of adenocarcinoma of oesophagus. Cancer 1989; 64: 526-530.
8. Johnston BJ, Reed PI. rising incidence of oesophageal adenocarcinoma : A 20 year retrospective review fro a single unit. Eur J Cancer Prev 1991; 1: 23-25.
9. Devesa SS. Blot WJ. Changing patterns in the incidence of oesophageal and gastric carcinoma in the United States. Cancer 1998; 83: 2049-53.
10. Jankowski JA, Wright NA. Molecular evolution of the metaplasia-dysplasia-adenocarcinoma sequence in the esophagus. Am J Pathology 1999; 154: 965-973.
11. Allum, Grittin. Guidelines for the management of oesophageal and gastric cancer. Gut 1999; 6: 39.
12. Dolan K, Walker SJ. Adenocarcinoma of the lower oesophagus and cardia are similar both epidemiologically and clinically. A population based study from 1974-1993. Gut 1977; 40(supp): A 37.
13. Sons HU. Cancer of the distal esophagus and Cardia. Incidence, tumours infiltration and metastatic spread. Ann Surg 1986; 203(2): 188-195.
14. Byrne JD, Mathews JM, Parry JM. Site distribution of oesophago-gastric cancer. J Clin Pathol 2002; 55: 191-194.
15. Antoninoli Da, Goldman H. Changes in the location and type of adenocarcinoma. Cancer 1982; 50: 775-781.
16. Blot W, Devesi S, Kneller. Rising incidence of adenocarcinoma of oesophagus and gastric cardia. JAMA 1991; 265: 1287-1289.
17. Jankowski et al. Gastro-oesophageal cancer : death at the junction. BMJ 2000; 321: 463-464.
18. Spechler SJ. Esophageal columnar metaplasia (Barrett's oesophagus). Gastroenterol Endosc Clin N Am 1997; 7: 1-18.
19. Nandurkar S, Talley NJ, Martin CJ, et al. Short-segment Barrett's oesophagus: Prevalence, diagnosis and associations. Gut 1997; 40: 710-715.
20. Spechler SJ. The role of gastric carditis in metaplasia and neoplasia at the gastro-oesophageal junction. Gastroenterology 1999; 117: 218-228.
21. Voutilainen M, Farkkila M, Mecklin JP. Chronic inflammation at the gastro-oesophageal junction (carditis) appears to be a specific finding related to Helibacter pylori infection and gastro-esophageal reflux. Am J Gastroentrol 1999; 94: 3175-3180.
22. Richardfs FM, Mckee SA, Rajpar MH, et al. Germline E-cadherin gene (CDHI) mutations predispose to familial gastric and colorectal cancer. Hum Mol Genet 1999; 4: 607-610.
23. Eastman Q, Grosschedl R. Regulations of LEF-1/JCF transcription factors by Wnt and other signals. Cure Opin Cell Biol 1999; 11: 233-240.
24. Crawford James A. The gastrointestinal tract - Robbins Pathologic Basis of Disease. Corran, Kumar, Collins, Saunders, Sixth Ed. 1999; 781.
25. Castellanos Rios E, Sitas F. Changing pattern of gastric cancer in Oxfordshire. Gut 1992; 33: 1312-1317.
26. Craanen ME, Dekker W. Time trends in gastric carcinoma: changing patterns of type and location. Am J Gastroentrol 1992; 87(5): 572-579.
27. Inove M, Tajuna K, Kitoh T, et al. Changes in histopathological features off gastric carcinoma over a 26 year period (1965-1990) J of Surgical Oncology 1993; 53: 256-260.
28. Hirayama T. Epidemiology of stomach cancer in Japan with special reference to the strategy for primary prevention. Jpn J Clin Oncol 1985; 14: 159-168.

EMPHYSEMATOUS PYELONEPHRITIS

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ABSTRACT: Emphysematous pyelonephritis is a life threatening necrotizing renal infection characterized by the production of gas. We present four cases, three of whom were females with diabetes, while one was non-diabetic male with urinary tract obstruction associated with urinary calculi. All four patients were managed aggressively initially with I/V fluids, antibiotics with correction of electrolytes and blood sugars. One patient was subjected to hemodialysis for acute renal failure and all four were latter subjected to emergency nephrectomy. Right kidney was involved in all the four patients. Though the mortality rate reported in 40-50%[1] primarily owing to sudden septic complications, we had no mortality although morbidity was high.

Key words: *Emphysematous, pyelonephritis, diabetes, nephrectomy, necrotizing renal infection, parenchymal gas*

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INTRODUCTION

Emphysematous pyelonephritis is an acute necrotizing renal parenchymal and perirenal infection caused by gas forming uropathogens[2,3] The pathogenesis is poorly understood, because the condition usually occurs in diabetic patients, and it has been postulated that the high tissue glucose levels provide the substrate for microorganisms such as E. coli, which are able to produce carbon dioxide by the fermentation of sugar[4]. In addition to diabetes, many patients have urinary tract obstruction associated with urinary calculi or papillary necrosis and significant renal functional impairment. The disease is considered rare and we presented four cases encountered during the last 4 years.

Report of cases

Case No. 1: A 65 years old female, non alcoholic, postmenopausal, known hypertensive and diabetic (insulin dependent) for last 5 years, presented with high grade fever, rigors and chills, vomiting and right side flank pain for last 5 days and oliguria for 2 days before admission. On examination the patient was febrile temp 102°F, ill looking, disoriented in grade III coma, BP of 90/60 mmHg, pulse 130/mt low volume with R/R of 38/mt. Auscultation of chest revealed B/L crepitus in lower zone. Abdominal examination revealed tenderness all-over the abdomen especially right flank with renal mass. Laboratory investigations showed Hb - 8.5 gm, TLC - 25,090, DLC-N90, L10, Platelet 90,000. Urea - 110, Creatinine 10.9 mg/dl, Na - 138, K - 6.8, Calcium - 9.6 mg/dl, blood sugar 445 mg/dl. Blood culture and urine culture were positive for E. coli and Klebsiella (Table 2). Urine exam - pus cells full field, proteinuria ++, sugar +++, ketoacidosis. X-ray chest P/A - mild (R) sided effusion. Ultrasonography showed enlarged right kidney with strong focal echoes. X-ray KUB - mottled gas shadows over the right kidney area. Non-contrast CT scan showed presence of intraparenchymal gas in the right kidney with enlarged, & distorted kidney with areas of necrosis. Immediately after admission, patient went into septic shock. Patient was resuscitated and put on ventilatory support with renal dose of dopamine. Patient was put on broad spectrum antibiotics and blood sugar controlled with plain insulin. After stabilizing the patient for 24 hours, patient was subjected to one cycle of hemodialysis. Emergency right side nephrectomy through 11th rib incision was performed and renal fossa copiously irrigated with and a large bore tube drain was kept in for 7 days. Operative findings recorded were enlarged and inflamed right kidney with adhesions to the ascending colon, duodenum, liver, psoas muscle and IVC. Chocolate coloured pus was seen in perirenal area with sloughing of renal parenchyma. Pus culture from the operative site revealed E.coli and Klebsiella. Pathology report revealed diffuse renal ischemia, with varying degree of glomerulosclerosis, arterionephrosclerosis with arterial and venous thrombosis with papillary necrosis. **Course and outcome:** - Patient was managed on ventilator for 3 days after surgery and all vital parameters were kept under near normal with one more hemodialysis given postoperatively on 1st POD and patients blood pressure maintained on dopamine (5ugm/kg/min). Postoperatively two units of A+ve blood was transfused. Patient started forming urine from 2nd POD. Patient was shifted from ICU on 6th POD. Rest of the postoperative course remained uneventful except wound infection. Patients creatinine level came down to 2.5 mg/dl on 10th POD and at the time of

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discharge on 21st POD, creatinine was 1.7 mg/dl (Table 2) and blood sugar fasting was 88 mg/dl on insulin. At 3 months of followup creatinine was 1.0 mg/dl with controlled blood sugar.

Case no. 2: A 58 year old female known diabetes (NIDDM) for last 2½ years on erratic treatment was admitted with high grade fever with rigors and chills, increased frequency of urine, flank pain with uncontrolled blood sugars of 5 days duration. On physical examination patient was febrile, temp 101°F, dehydrated, irritable, pulse 126/mt low volume, BP 100/70 mmHg with tenderness in right flank and right hypochondrium Laboratory studies showed HB - 10.5 gm%,TLC 18,680, DLC - N88, L08, E4, urine routine - Alb +, sugar +++, with pus cells full field. Urine and blood cultures showed E. coli. Urea - 90 mg/dl, creatinine 2.5 mg/dl, blood sugar (F) 375 mg/dl (Table I). X-ray KUB was inconclusive as was ultrasound. Plain CT scan showed presence of parenchymal gas in the right perirenal and intraparenchymal region. Patient was resuscitated with IV fluids, broad spectrum antibiotics and control of blood sugar with regular insulin and emergency right nephrectomy with 11th rib incision was performed within 8 hours of admission. Operative and pathological findings were same as in case no. 1. Pus culture from operative site showed E.coli. Patient did well postoperative except wound infection, which needed secondary suturing. Creatinine level stabilises to 0.9 mg/dl from 2nd POD and at the time of discharge remained 0.9 mg/dl. At 3 month of follow up, blood sugar were 78 mg/dl and creatinine of 0.9 mg/dl.

Case no. 3: A 62 years old female, known diabetic for last 6 yrs (NIDDM), was shifted from medical ward with H/O fever, rigors and chills, pain right flank and right renal mass with uncontrolled blood sugars for last 7 days. On physical examination patient was stable, febrile 104°F, BP 130/90 mmHg, pulse 100/ mt regular. There was tenderness in the RHC and RLR and a right renal mass which was tender. Laboratory studies revealed Hb-9gm,TLC-14,300, DLC- N80, L10 E06, M04, urea 100 mg/dl, creatinine 3.5 mg/dl, blood sugar 410 mg/dl, Na 140, K 4.5, urine exam - pus cells 10-15, sugar ++, Alb +, urine culture- E.coli, and blood culture was negative.X-ray KUB was inconclusive. Ultrasound reported enlarged right kidney with mixed internal echogenicity suggestive of renal mass (RCC).CT scan reported presence of intraparenchymal and pararenal gas in the right kidney with areas of necrosis. In medical ward patient was managed with IV fluids, broad spectrum antibiotics as per culture & sensitive of urine and blood sugar were controlled by giving human insulin S/C and patient also received two units of B+ve blood transfusion. Immediately after shifting the patient to urology deptt, an emergency right nephrectomy was performed through 11th rib incision. Operative findings reported were enlarged and inflamed right kidney, pararenal and perirenal abscess, with sloughing of the renal parenchyma with areas of necrosis in between. There were lot of adhesions with the surrounding structures. Pathology report was same as in case 1. Pus culture from operative area reported growth of E.coli. Postoperative period was uneventful. Drain was removed on 5th POD. Creatinine was 1.5 mg/dl and blood sugar (F) 100 mg/dl on 3rd POD. Except for wound infection and right basal pneumonia, patient recovered well. At the time of discharge, patients creatinine was 1.5 mg/dl and at 3 month of follow up it was 1.0 mg/dl.

Case no. 4: A 50 yr old male non-diabetic, non-hypertensive was admitted with history of high grade fever with rigors and chills, pain right flank and right renal mass of 14 days duration. Past history, patient had urethral stricture since 1986 and was subjected to optical urethrotomy in 1987, 1991 and 1995. Finally in 1998, patient had undergone cystolithotomy with urethroplasty and since then was doing well. On physical examination patient was found febrile, temp 102°F, pulse 120/mt, BP 110/70 mmHg, grossly pale and tachypneic. Abdomen was tender on right side (RHC + RLR) with a palpable renal mass measuring about 10 x 14 cm, slight tender with subcutaneous crepitus felt in the right renal area over the mass. Chest examination revealed right side pleural effusion. Laboratory studies revealed Hb 6.5 gm, TLC- 25,000, DLC-N90,L08,E02, platelet- 90,000, urea 90 mg/dl, creatinine 2.5 gm/dl, Na 134, K 4.9, calcium 9.6, blood sugar fasting 72 mg/d. Urine examination revealed pus cells full field with 10-20 RBC, sugar nil, Alb ++, urine culture- E. coli, and blood culture was negative. Ultrasound reported right renal mass with altered echogenicity. X-ray KUB showed presence of renal calculi with mottled gas shadows in the right kidney area (Fig..1). X-ray chest PA view showed right lower zone effusion.. Thoracocentesis reported transudate with no evidence of any malignant , inflammatory cells or tuberculosis. CT scan reported presence of gas in the intraparenchymal, pararenal and in the collecting system (Fig .2), patient was managed with IV fluids, broad spectrum antibiotics, and received 3 units of B+ blood transfusion for 2 days and then patient was subjected to emergency right nephrectomy. Operative findings reported were enlarged and grossly inflamed right kidney, para and perirenal chocolate coloured abscesses with dense adhesions to ascending colon, duodenum, liver, pleura, IVC and psoas muscle, with large renal calculi > 3 cms in dia at PUJ region. Pathology reported obstructive features superadded with inflammation. Pus culture from the operative site growed E. coli and klebsiella. Postoperative period was uneventful. Creatinine on 2nd POD was 0.9 mg/dl and drain was removed on 5th POD.

Table 1: Historical and admission data of 4 cases

1. Mean age (yrs)	58.7 yrs (50-65)
2. Total no. of pts	4 patients
3. Male : Female ratio	1:3
4. Medical history	
UTI	1
Stone (no)	1
Diabetes (no)	3
5. Symptom duration (days)	
Mean S. duration	7 days (5-14 days)
Fever and rigours (no)	4
Pain (no)	3
Vomiting (no)	3
Renal mass (no)	3
6. Admission diagnosis (no)	
Diabetic ketoacidosis	1
Diabetes out of control	3
UTI and sepsis	1
7. Admission physical examination (no)	
Temp 101% (no)	4
Cast vertebral angle tenderness (no)	4
Hypotension(BP < 100 mmHg)	1
Subcutaneous air	2
8. Mean admission laboratory studies	
Hb (gm)	8.6 gm (6.5 - 10.5)
WBC count	20,875 (14,300 - 25,090)
Glucose (F)	327 mg/dl (78-440)
9. Percentage sensitivity of various investigations for presence of gas	
Plain KUB	75%
USG	25%
Non-contrast CT	100%

Except for wound infection and right basal pneumonia, patient recovered well. At the time of discharge and at 3 months of followup creatinine was 0.9 mg/dl.

DISCUSSION

The Pathogenesis of emphysematous pyelonephritis appears to be poorly understood. It has been postulated that pyelonephritis developing in the diabetic kidneys is followed by some unknown stress or event that induces diffuse renal ischemia. The resultant low oxygen tension induces anaerobic metabolism. Escherichia coli, klebsiella pneumonia and proteus mirabilis, which are facultative anaerobes, then are able to ferment glucose to lactate and carbon dioxide. It seems more reasonable to postulate that impaired host response caused by local factors, such as obstruction, or a systemic condition, such as diabetes, allows organisms with the capability of producing carbon dioxide to utilizes necrotic tissue as a substrate to generate gas in vivo. This emphysematous pyelonephritis should be considered a complication of severe pyelonephritis rather than a distinct entity. All of the documented cases of emphysematous pyelonephritis have been adults[5]. Juvenile diabetic patients do not appear to be at risk. Women are affected more often than men and right kidney is involved more often than left kidney[1]. Almost all patients display the classic triad of fever, vomiting and flank pain[4]. Pneumaturia is absent unless the infection involves the collecting system. Results of urine cultures are invariably positive, E. coli been the most common uropathogen, followed by klebsiella and proteus. The clinical findings in our patients are similar to those generally reported in the literature[2, 6].

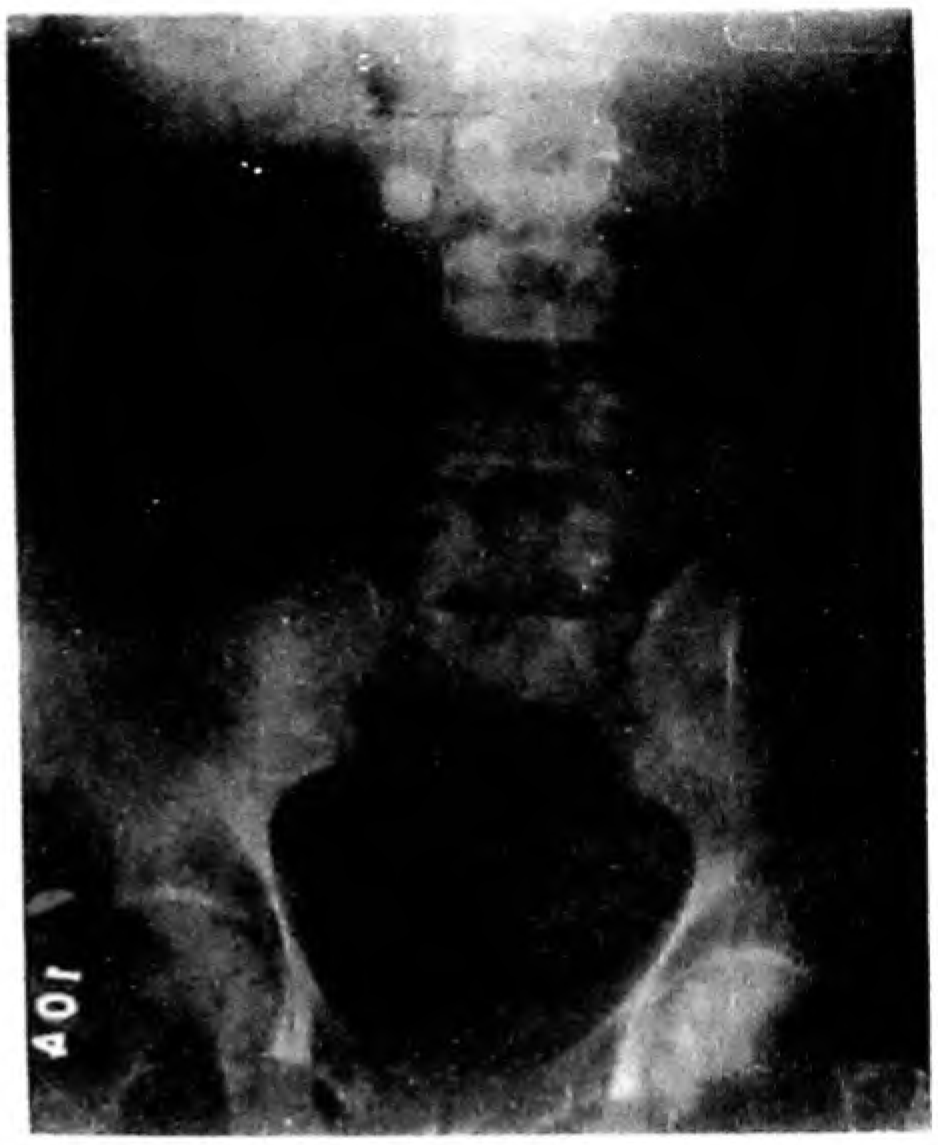

Fig. 1

Table 2: Results of urine culture, pus culture and blood culture of 4 patients

Case no.	Urine culture	Pus culture taken from lesion at the time of surgery	Blood culture
1.	E.coli + Klebsiella	E.coli + Klebsiella	E. coli
2.	E.coli	E.coli	E.coli
3.	E.coli	E.coli	Negative
4.	E.coli	E.coli + Klebsiella	Negative

Table 3: Summary of creatinine levels at admission, discharge and at 3 months of followup of 4 patients-

Case no.	Patient age yrs	creatinine levels Admission	Discharge	3 month follow up
1	65 F	10.9 mg/dl	1.7 mg/dl	0.7 mg/dl
2	58 F	2.5 mg/dl	0.9 mg/dl	0.9 mg/dl
3	62 F	3.5 mg/dl	1.5 mg/dl	1.0 mg/dl
4	50 M	2.5 mg/dl	0.9 mg/dl	0.9mg/dl

As reported by Schainuck et al [4], the production of carbon dioxide is responsible for the appearance of gas on x-ray examination. Furthermore, the poor blood flow exacerbates the situation by inhibiting antibiotic perfusion to the kidney. Thomas E, et al[1] found that entire kidney was involved and no real sparing of renal parenchyma exists to allow partial nephrectomy, as was seen in our cases also. Patients with emphysematous pyelonephritis involving the right kidney reportedly are at greater risk for death[2], owing to the increased difficulty of removing an enlarged and inflamed kidney on the right side with inferior vena cava, liver and duodenum in such close proximity.

Radiographically, the plain film of the kidney, ureters and bladder (KUB) is the best study to screen diabetes for air in the renal fossa. However, this finding is often mistaken for bowel gas (Fig. 1). A crescentic collection of gas over the upper pole of the kidney is more distinctive. As the infection progresses, gas will extend to the perinephric space and retroperitoneum.

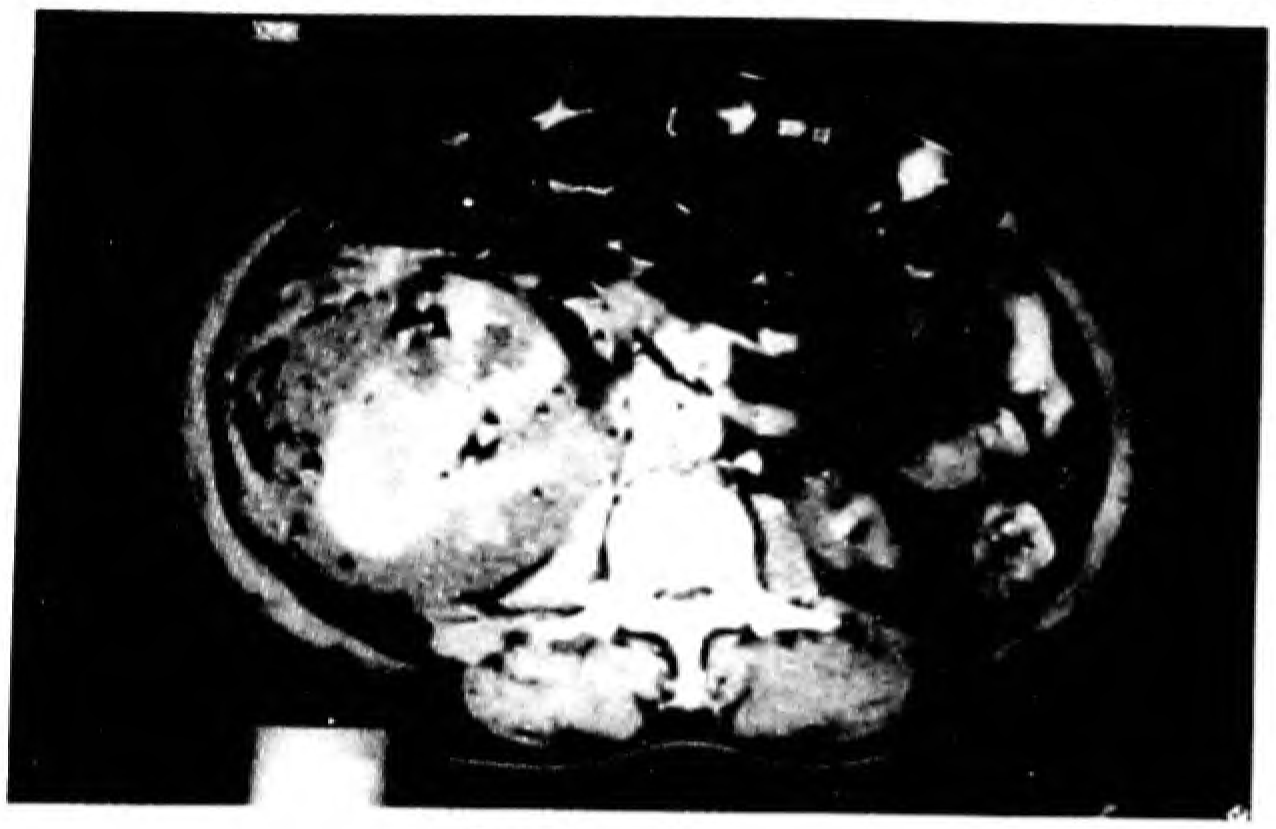

Fig. 2

Excretory urography is rarely of value, since the affected kidney usually is non-functioning or poorly functioning. Because of the significant risk of contrast nephropathy in critically ill, dehydrated diabetic patients with abnormal renal function, non contrast CT scan[7] is more precise, with its high resolution and lack of need for contrast material and is 100% diagnostic in the presence of suspicious plain films, as was noted in all our cases (Fig. 2). Ultrasound is usually less informative and usually demonstrate strong focal echoes suggesting the presence of intraparenchyma gas[8,9]. We performed ultrasonography on all patients and was diagnostic only once. Renal scan should be performed to assess the degree of renal impairment and the contralateral function.

Management of patients with emphysematous pyelonephritis has been the topic of many reports. Most investigators agree that nephrectomy is the preferred management[2,6]. Emphysematous pyelonephritis is a surgical emergency and most patients are septic. Fluid resuscitation and broad spectrum antibiotics are essential. If the kidney is functioning, medical therapy can be considered. Nephrectomy is recommended for patients who do not improve after a few days of therapy[10]. If the affected kidney is non-functioning and not obstructed, nephrectomy should be performed, since medical treatment alone is usually lethal especially in diabetic patients. If the kidney is obstructed, catheter drainage must be instituted. If the patients condition improves, nephrectomy may be deferred pending a complete urologic evaluation. Although there are isolated case reports of retention of renal function after medical therapy combined with relief of obstruction, most patients require nephrectomy[11]. We have used nephrectomy in all patients and agree that nephrectomy is the preferred treatment management, as delayed nephrectomy only jeoparadizes the chances for survival. Even with aggressive management, the mortality rate in Thomas E et al[1] study was 42%, while Michaeli et al[2] reported survival of approximately 90%. We had no mortality in our four patients.

CONCLUSION

In summary, emphysematous pyelonephritis is a variant of pyelonephritis that must be respected for its malignant potential. Diagnosis is confirmed by gas in the renal parenchyma or perinephric space either by a plain KUB or noncontrast CT scan. High suspicion should be made in patients with pyelonephritis associated with diabetes or with urinary obstruction. Appropriate medical management and stabilization of the patient should be attempted but immediate nephrectomy must not be delayed. The process continues to have a 40% - 50% mortality rate primarily owing to sudden septic complication. At operation a high 11th or 10th rib incision should be made to allow easy access to the renal vessels, which should be secured first. The area should be irrigated copiously, a large drain should be placed. Our experience with four patients reveals that rapid improvement ensures and no significant deterioration of renal function occurs if immediate nephrectomy is performed.

References

1. Thomas E, Ahlering, Stuart D. Boxd, Catherine L. Hamilton et al. Emphysematous pyelonephritis : A 5 year experience with 13 patients .J Urol 1985; vol 134: 1086-1088.
2. Michaeli P, Mogle P, Perlberg S, Heiman S and Caine M. Emphysematous pyelonephritis. J. Urol 1984;131:203.
3. Schultz EH; Jr and Klorfein EH. Emphysematous pyelonephritis. J Urol 1962;87:762.
4. Schainuck L.I, Faity R and Cutler RE. Emphysematous pyelonephartis. A new case and review of previous observations. Amer J Med 1968;44:134.
5. Hawes S et al. Emphysematous pyelonephritis. Infect Surg 1983;2:191.
6. Freiha, F.S, Messing EM and Gross DM. Emphysematous pyelonephritis. J.C.E. Urol June 1979;18:9.
7. Kim DS, Woesner ME, Howard TF, et al. Emphysematous pyelonephritis demonstrated by computed tomography. Am Roentgen Ray Soc 1979;132:287.
8. Brenbridge AN, et al. Renal emphysema of the transplanted kidney. Sonographic appearance. AJR 1979;132:656.
9. Conrad MR, Bregman R, Kelman WJ. Ultrasonic recognition of parenchymal gas. AJR 1979;132:395.
10. Elder JS. Xanthogranulomatous pyelonephritis and gas forming infections of the urinary tract. AUA Update 1984;111:2, Lesson 31.
11. Hudson MA, Weyman PJ, Vander Vliet AH, et al. Emphysematous pyelonephritis: Successful management by percutaneous drainage. J Urol 1986;136:884.

LAPAROSCOPIC CHOLECYSTECTOMY: AN ANALYSIS OF 100 CASES

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Abstract: *The study was conducted in the department of surgery, SMHS hospital Srinagar, on 100 patients admitted by the surgical unit for cholecystectomy from June 1999 – October 2001. There was no criteria regarding age and sex of patients. However, care was taken not to include patients with associated pathology like choledocholithiasis, GB malignancy or severe cardiovascular / pulmonary disease and major bleeding disorders. Indication for surgery included chronic calculus cholecystitis 78, acute calculus cholecystitis 12, mucocoele 5 gallbladder polyps 2, empyema gallbladder 2, and a calculus cholecystitis 1, 9 patients were converted to open procedure giving the conversion rate of 9 %. There was one major complication (Right hepatic duct injury) and few minor complications. The mean operative time was 57.43 min with range from 15- 220 minutes. The average hospital stay was 3.25 days and range from 2- 7 days, no death occurred in this series. Our results suggest that laproscopic cholecystectomy is essentially a safe procedure with low morbidity and mortality rates.*

KEY WORDS: *Laparoscopic Cholecystectomy- Complications*

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Cholelithiasis is one of the most common diseases of western countries with a prevalence of 5-221 % and in western world it is the commonest surgical procedure in abdomen[2]. An estimated 20 million people in United States harbour gall stones and approximately 500,000 Cholecystectomies are done annually[3, 4]. In our part of the country also this disease is very common[5]. The basic technique of conventional Cholecystectomy by Carl langenbuch of Berlin (1882) remained unchanged for about a century when Philippe Mourets in Lyon France did the first laparoscopic surgery in March 1987[6,7,8,9].

Laparoscopic surgery provides quicker and comfortably recovery with rapid return to work by reducing the trauma of operative access. We report here our results during Ist year of laparoscopic cholecystectomy in SMHS Hospital.

MATERIAL AND METHOD

100 patients with symptomatic gall stones/ polyps under went laproscopic cholecystectomy from june 1999 – Oct 2001. The series included 76 females and 24 males. In all the patients diagnosis was confirmed by USG. Routine preoperative investigations included full blood count, LFT,KFT, electrolyte and clotting profile.

On the day before surgery an informed consent was taken for the procedure after explaining the patient the advantages and potential complications of the procedure. Patient was also told that in the event of intraoperative difficulties, the procedure might be converted to open cholecystectomy. Soft diet was prescribed to patients on the evening before the surgery and preoperative enema was prescribed in the late evening and morning before surgery. ceftrioxone 1 gm was given at the time of induction and the patients were directed to void the bladder before surgery. All the patients were operated under general anaesthesia nasogastric tube was inserted in all the patients at the time of induction and removed at the end of operation. The operative procedure was conducted in the manner described by others[10,8] Intra operative cholangiography was not performed in any case.

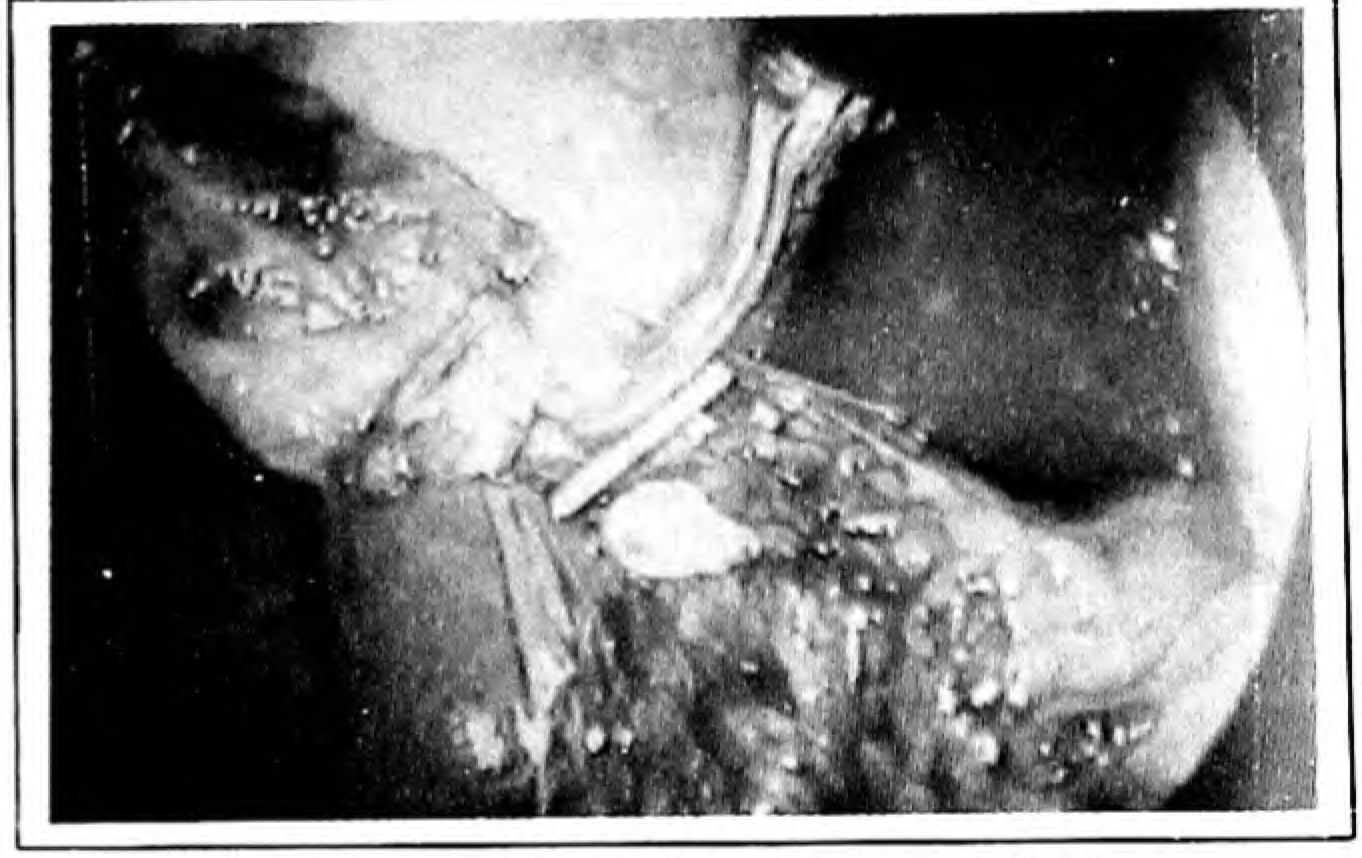

Fig. 1 Cystic duct being clipped and cut with scissors

Normal diet was started on the second postoperative

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day and the majority of the patients were discharged on the 2nd postoperative day. All patients were reviewed on OPD basis with in a week of discharge.

RESULTS:

In the study majority of the patients (58%) were in 3rd and 4th decade of life, (35% were in the age group of 31 to 40 years and 23% in the age group of 41 to 50 years).Mean age was 41 years with range from 16 to 85 years. Females dominated the number with 76 femalesand 24 males. Male :female ratio was 1 : 3. The operative findings are tabulated below:

The procedure was converted into open cholecystectomy in 9 patients and the reasons for conversion are summarized in Table 2..the mean operative time was 57.43 min and range from 15 – 220 minutes the hospital stay ranged from 2-7 days with average od 3.25 days. Conversion to open procedure incresased hospital stay in some patients

Table 1: Distribution of Cases According to Operative Findings

Operative findings	NO	%age
Adhesion free or minimal soft omental adhesions	69	69
Dense visceral or omental adhesions	31	31
Obliteration of Calot's Triangle by adhesions and edema	17	17
Thin walled Gallbladder	86	86
Thick walled and inflamed Gallbladder	12	12
Dialated CBD	2	2
Choledocholilithiasis and worm in CBD	1	1
GB Malignancy	2	2
Mucocoele	5	5
Empyema	2	2
Cholecysto- Gastric Fistula	1	1
GB polyp	2	2
No Stone (Acalculus cholecystitis)	1	1
Fatty Liver	1	1
Acessory Cholecystohepatic duct	2	2
Anomolous cystic artery	1	1
Situs inversus	1	1

TABLE 2: Distribution of Cases According to Reasons of Conversion

REASONS FOR CONVERSION	No.	%age
Acute cholecystitis with dense adhesions around oedematous Gall bladder with unclear anatomy	1	1
Acute Cholecystitis with cholecystogastric fistula and dilated CBD	1	1
Acute Cholecystitis with accessory cholecystohepatic duct with unclear anatomy	1	1
Mucocoele with adherent right hepatic duct which was damaged during dissection	1	1
GB Malignancy with secondaries liver	1	1
Mass at the fundus of gallbladder (Malignant)	1	1
Dilated CBD with choledocholithiasis and dead worm in CBD	1	1
Anomolous cystic artery injury.	1	1
GB Suitated on Left Side (Situs Inversus) which was not documented preoperatively	1	1

COMPLICATIONS: The complications were divided into intraoperative and post operative complications which were further sub divided into major and minor complications. The various complications seen in our study are tabulated below.

TABLE 2: Distribution of Cases According to Intraoperative Complications

COMPLICATIONS	No	%age
Major Complications		
Right hepatic duct injury	*1*	*1*
Minor Complications		
BLEEDING		
Bleeding from cystic artery	7	7
Bleeding from liver bed	6	6
Bleeding from epigastric port	2	2
Bleeding from right lower port	1	1
LIVER INJURY	1	1
GB PERFORATION WITH SPILLAGE OF BILE	21	21

RETURN TO WORK: Time taken by the patient to return to work and pre-operative activities from the day of operation was determined

TABLE 4: Distribution of Cases According to Time Taken to Return to Normal Activity. (Lap. Chole Group)

TIME IN WEEKS	No.	%age
1-2	62	62
3-4	28	28
5-6	1	1

TABLE 2: Distribution of Cases According to Time Taken to Return to Normal Activity (Conversion Group)

TIME IN WEEKS	No.	%age
3-4	2	2
5-6	5	5
7-8	2	2

Discussion and Conclusion

Laparoscopic Cholecystectomy is rapidly replacing the conventional open cholecystectomy for treatment of cholelithiasis due to its obvious benefits to the patient. It is now offered to "all the comers" with a success rate of 99[11]%. This technique of minimum access surgery has gained wide acceptance because of high quality of results obtained in various series[11,12]. The conversion rate in our series has been (9%) which is similar to the conversion rate (0.08% to 14%) reported in world literature[13] .The reasons for conversions were (a) unclear anatomy with difficult dissection at Calot's triangle in acute cholecystitis (3%) (b) GB malignancy (2%) (c) dilated CBD stone with choledocholithiasis (1%) (d) mucocoele with adherent right hepatic duct (1%) (e) Anomolous cystic artery injury (1%) (f) situs inversus (1%)

Injury to bile duct causes the most serious morbidity associated with laparoscopic cholecystectomy and is considered to be a major complication.our series, the incidence of bile duct injury was (1%). Which is similar (0-2%) to the incidence in published series of world literature This patient had mucocoele of gallbladder and right hepatic duct was completely adherent to gallbladder wall. Right hepatic duct was damaged during dissection with diathermy hook, so conversion was decided. Right hepatic duct was sutured over T-tube with interrupted 3-0 vicryl.

A port site metastases rate of 6.7% has been reported after laproscopy which is a higher figure as compared to metastasis in laprotomy line after open surgery. In one of our patients with gallbladder malignancy, no port site metastasis was seen upto a period of 6 months follow up. The other patient did not report for follow up:

Intraoperative bleeding was encountered in 17 patients. In 7 patients there was bleeding from cystic artery which was cut accidentally (2) or there was an early division of cystic artery into anterior and posterior branches which were not recognised (5) while anterior branch was secured, the posterior branch continued to bleed . Bleeding was easily controlled by applying a clip or using diathermy after irrigation and suction. Six patients had ooze from liver bed which was controlled by pressure and diathermy. In one patient bleeding due to accidental liver laceration by the grasper was controlled by pressure and diathermy.

One patient in our series drained 400 ml of bile on Ist Postoperative day and on second postoperative day again same amount of bile was drained. However, it gradually decreased to 100ml / day upto 6th postoperative day and then upto 50ml / day till 8th postoperative day. Tube drain was clamped after 8 days for 24 hours. Patients did not get any pain, fever or jaundice and ultrasound was done which was normal with no collection in abdomen or pelvis. However the cause of bilary leak could not be established and the drain was removed on the next day. Another patient in series drained 200 ml of bile in Ist 24 hours after surgery, 50 ml on 4th and 5th postoperative days and 10 ml on 6th postoperative day and ultrasonography did not reveal any residual collection and the drain was removed on the same day. The mean hospital stay in our series was longer as compared to the reports in literature. Most of the patients who had undergone laparoscopic cholecystectomy were fit for discharge on second postoperative day only. The patients were delibrately discharged on 3rd or 4th postoperative day depending upon the distance from the hospital as there is a paucity of domicilliary of nursing and hospital based healthcare facilities in the peripheral areas of our valley. Thus the benefit of early discharge after laparoscopic cholecystectomy was less evident in our series as compared to the reports in the world literature.

One of the main advantages of laparoscopic cholecystectomy is the potential for the patients to return to work much earlier than after conventional open cholecystectomy. In a study conducted by Mc. Intyre et al an early return to work with mean time of two weeks has been observed. In our series, it was observed that educated office going people did resume their work as early as 2-3 days; the range has been between 1-6 weeks and 29 patients resumed routine work after 3 weeks.. The reason was that there is a general perception in the community that removal of gallbladder by whatever means being a major operation needs a longer perioid of convalescence for recovery. It is also possible that some patients use the operation as an excuse to gain a few extra weeks to escape from their work, especially if they are being paid there remuneration during the leave period Since we are entering an existing new era of laparoscopic surgery where there is a continuos public and professional demand for minimal access surgery, so it is our moral responsibility to benefit our patients from this new technology. However, the patients safety must be paramount and it is necessary to ensure that all the surgeons receive the highest quality training so that the techniques are applied appropriately.

Our series indicated an overwhelming patient satisfaction in particular refrence to postoperative pain, hospital stay, return to normal activity and from cosmetic point of view.

The conclusion drawn from our study is that laparoscopic cholecystectomy is essentially a safe procedure with low morbidity and mortality rates. In addition complications of laparoscopic cholecystectomy can be minimised by maintaining "low threshhold" for conversion to laprotomy. It must be remembered that conversion should never be considered as a failure, but in fact, is a mark of prudence

References:

1. Neugebauer E., H.Triodl, W. Spangenberger et al. Conventional versus. Laproscopic Cholecystectomy and the randomized controlled trial. **Br. J.Surg, Vol.78, February, 150-154.**
2. R. C. G Russel, Norman S Williams, Cristopher J K Bulstrode 23rd Edition, Baily And Love's Short Practice Of Surgery -974.
3. David C. Sabiston, Fifteenth edition: Text Book Of Surgery – The Biological basis of modern Surgical Practice – 791,1132,1126
4. Moosa A..R. David W. Easter, Eric Van Sonnenberg et al. Laparoscopic Injury to Bile Duct-A Cause for Concern. Ann. Surg.Vol.215, No.3. 203-208 March 1992
5. Kapoor K.L, Ahmad S. P.L Kariholu et al : Benign Gall bladder disease – Indian Jr. of Surgery 1984 341-344
6. Baev.St, Pozarliev.T, Todorov G.T et al Laparoscopic Cholecystectomy: 700 Çonsecutive cases Int.Surg.1995;80:296-298.
7. Brown TH, M.H Irving:. Introduction to Minimal Acess Surgery: 1-3, BMJ Publishing Group 1995..
8. Nathanson L.K , Shimi. S, Cuschieri. A et al Laparoscopic Cholecystectomy: the Dundee Technique. Br.J. Surg. 1991, Vol. 78, February, 155-159.
9. Schirmer Bruce D, Janet Dix, Pa-C,Stphen B.Edge, Matthew JH et al Laproscopic Cholecystectomy in obese patients Ann. Surg. Vol 216 No.2 146 – 151 August 1992
10. Schimmer BD, Edge SB, Dix J et al Laproscopic cholecystectomy. Ann. Surg . 1991; 213 : 665-676
11. Meshikhes Abdul Waheed, Samir AL Dhurais, D Bhatia et al Laproscopic cholecystectomy the Dammam central hospital experience Int .Surg 1995; 80: 102-104.
12. Reddick EJ, Olsen D, Spaw A, et al: Safe performance of Laproscopic cholecystectomy. The Amer. Jour of Surg

1991;161:377-381.

13. Zucker KA, Biley RW laproscopic guided Cholecystectomy. A Plea for enthusiasm. Amer. J of Surg 1991; 161:36-44
14. Spawat, Reddick EJ, Olsen DO. : Laproscopic laser cholecystectomy : An analysis of 500 procedures. Surg Laprosc Endosc 1991:1: 2-7.
15. Grace P, Quershi A, Darzi A et al : Laproscopic cholecystectomy A hundred consecutive cases Jr. Med J 1991 ;8412-14.
16. Braghetto I, J Bastias, A Cs endes et al : gall bladder carcinoma during Laproscopic cholecystectomy: Is it associated with bad prognosis? Int Surg 1999 ;84: 344 –349
17. Wolfe BM, Gardiner BN, Leary BF et al Endoscopic cholecystectomy .An analysis of complications Arch Surg.1991; 126:1192-8
18. Peck JJ, Endoscopic cholecystectomy : An analysis of complications -See comment Arch Surg.1991;126:3-12.
19. McMahon AJ, G.fullarton JN Baxter et al : bile duct injury and bile leak in Laproscopic cholecystectomy Br.Jr.Surg,1995 82:307-313.

SAUVE-KAPANDJI PROCEDURE

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Abstract: *Recently the Sauve-Kapandji procedure has become the most popular method usedfor the treatment ofvarious DistalRadioUlnar Joint disorders.However some complications,especiallypain over proximal ulnar stump due to instability have been reported in more recent studies. To prevent this pain,we have used a modified Sauve-Kapandji procedure,using Extensor Carpii Ulnaris tendon to suspend the proximal ulna stump.*

We report here 10 patients ofDRUJ disorders treated by this modified procedure between Jan.2002 and Jan.2003,at an average follow up period of 33 weeks,ranging from 16-50 weeks.The average age at the time of procedure was 33.5 years. There were 2 cases of chronic dislocation of DRUJ, 5 cases of post traumatic DRUJ arthritis and 3 cases of malunited fractures of distal radius. The distal end of proximal ulna was stabilized with a sling created using ulnar half slip of extensor carpii ulnaris tendon. Functional results were evaluated with Mayo wrist score,(2) Forearm rotation and X-rays.

Fusion of DRUJ was achieved in all cases by Two months. Excellent results were achieved in 5 cases, Good in 4 and fair in 1 case. All cases gained increase in range of forearm rotation. None of our patients had proximal ulnar stump pain or instability. There was no closure of pseudoarthrosis in our study. All of our patients were happy with the cosmetic appearance of the wrist.

Key words: *DRUJ disorders, DRUJ fusion, Sauve-Kapandji procedure, Mayo wrist score, ECU sling, Ulnar proximal stump.*

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Introduction:

The distal radioulnar joint is important in providing support to the ulnar side of the wrist.DRUJ disorders cause pain and limitation of forearm rotation. It leads to severe limitation of hand function and inability to perform activities that require forearm rotation. In 1936, Sauve and Kapandji(1) described an operation consisting of DRUJ fusion, creation of a pseudoarthrosis proximal to the fusion, has become a popular procedure for the treatment of various disorders of DRUJ. It eliminates pain, restores forearm rotation and stabilizes the DRUJ. However some complications, especially pain over the stump of the ulna, have been reported in more recent studies. To prevent the occurance of stump pain due to the instability of the ulna several modifications have been proposed (14,15,17). We report here the surgical technique and the results of our modified Sauve-Kapandji procedure for the treatment of DRUJ disorders.

Materials and Methods:

Between Jan.2002 and Jan.2003, 10 patients of DRUJ disorders were treated with Sauve-Kapandji procedure together with creating a sling using ulnar half of ECU tendon to stabilize the proximal stump of ulna, at Govt.Hospital for Bone and Joint surgery,Srinagar. All patients had wrist pain and a limitation in forearm rotation due to osteoarthritic change or an incongruity of the DRUJ. All the 10 patients were available for follow up at an average follow up period of 33 weeks,ranging from 16-50 weeks. The average age at the time of procedure was 33.5 years, ranging from 20-47 years. There were 6 males and 4 females. 7 procedures were performed on the right hand and 3 on the left hand. Four were manual workers, three were non manual workers and three were housewives. The procedure was indicated in those patients with DRUJ arthritis,dislocation or destruction and had high functional demands with severe unrelenting pain at the DRUJ. There were 2 cases of chronic dislocation of DRUJ, 5 cases of post traumatic arthritis of DRUJ and 3 cases with malunited fracture of the distal radius.

Pre-operatively, the patients were evaluated with Mayo wrist score(2). forearm rotation and radiologic examination.Fig 1. Patients were investigated before undergoing surgery as a routine.

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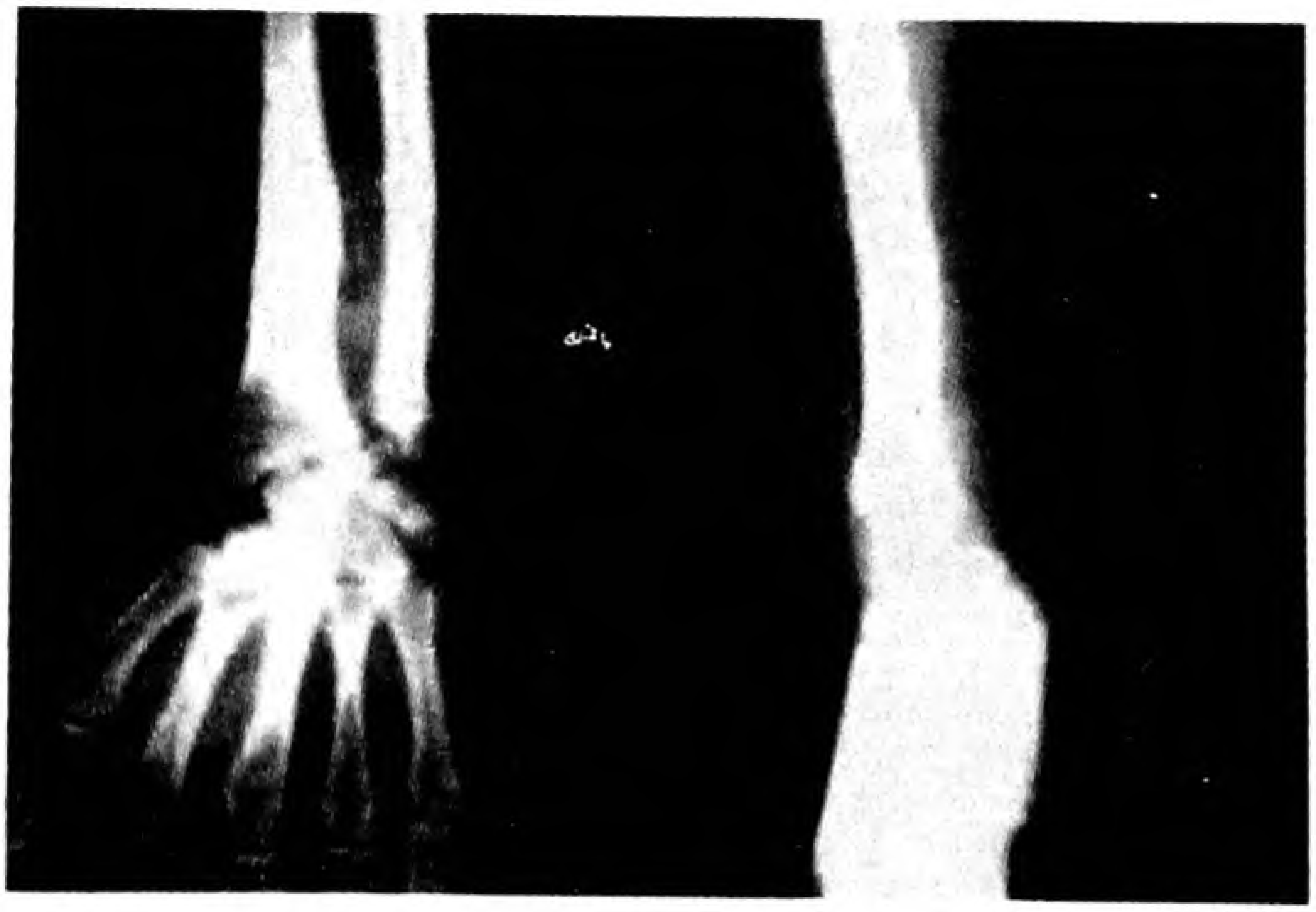
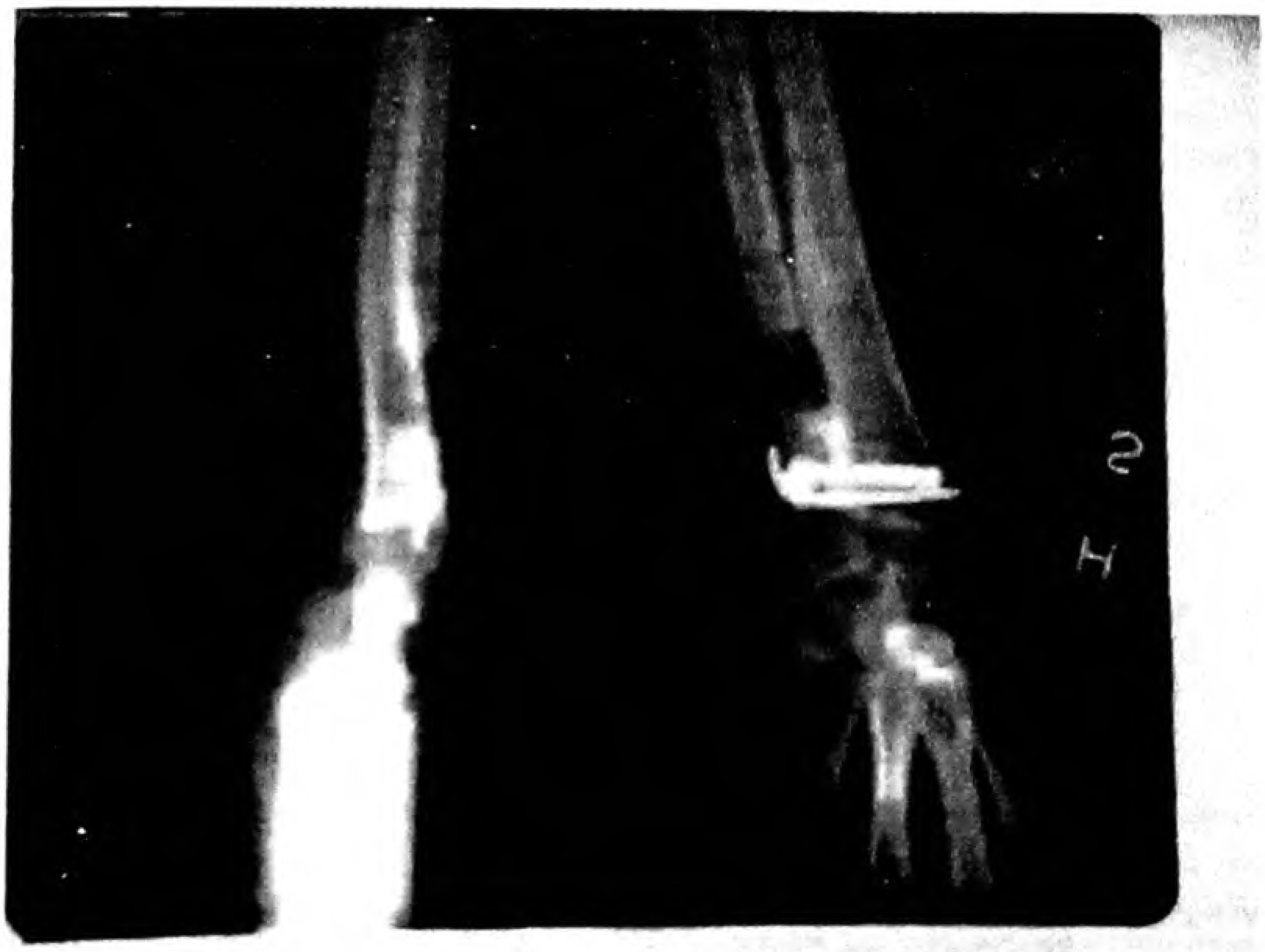
Radiographs of wrist:

PRE-OP:- (AP/LAT), showing Post-traumatic degeneration of DRUJ.



Post-op X-ray (AP/LAT) showing arthrodesis of DRUJ and Pseudoarthrosis site.



Technique:

The procedure was carried out according to Sauve-Kapandji method. The DRUJ was exposed through a dorsal incision. The DRUJ was fused with a Screw and a 'K' wire in some cases. A pseudoarthrosis was created proximal to arthrodesis by resecting a centimeter segment of Ulna. The resected bone was used as a graft to fill the space in DRUJ. The distal end of proximal ulna was stabilized with a sling created using the ulnar half of ECU tendon. The tendon was split along the midline from the musculotendinous junction to the insertion of tendon on Pisform. The ulnar half was transected near the distal attachment of the tendon. A 3.5mm drill hole was made proximal to the distal end of ulnar shaft in a dorsal to volar direction. The proximally based ulnar half of ECU tendon was passed through the tunnel from volar to dorsal direction and was sutured back to the ulnar half of the tendon under appropriate tension in the neutral rotaion of forearm. Pronator quadratus was released form the distal ulna and reattached to the radial half of the ECU tendon in the area of resection. After confirming the tension of the transferred tendon during forearm rotation, the wound was closed . An above elbow plaster splint in neutral forearm rotation was applied.

Post-Operative:

The patients were instructed to keep the hand elevated for 24 hours to prevent oedema and stiffness. The patients were given antibiotics intravenously and analgesics. The sutures were removed on the fourteenth day and an above elbow plaster cast was applied. At suture removal patients were assessed for wound condition,pain and radiologic examination was done. Plaster was removed at 6 weeks postoperatively, and the patients were advised intermittent gentle range of motion of wrist,elbow and shoulder. Muscle strengthening and weight carrying excersices began when radiological union of arthrodesis was seen.

The clinical results at final follow up were assessed by mayo wrist score, forearm rotation and radiological examination.

Results:

All cases had poor mayo wrist score and poor forearm rotation with average of 33.2 degrees of pronation and average supination of 29.6 deg. The average grip strength was 10.4 kg. Radiological union of the DRUJ arthrodesis was seen in all cases by two(2) months. At an average follow up of 33 weeks, the functional results rated by Mayo wrist score(2) were Excellent in 5, Good in 4 and Fair in One. All cases gained increased range of forearm rotation with 72.6 deg. Of pronation and average of 70.8 deg.of Supination. No patient complained of pain over the proximal ulnar stump or feeling of insecurity of the bone. None of the patients had closure of pseudoarthrosis. The average grip strength at follow up was 23.3kg. All the patients returned to work or participated in pre injury household, recreational or leisure activities at an average of 68.2 days after the operation. All patients were pleased with the procedure in terms of pain and cosmetic appearance of the wrist.

Discussion:

In the treatment of DRUJ disorders, the aim is to relieve pain, correct deformity and restore forearm rotation. Several surgical procedures have been reported for the treatment of DRUJ disorders and the DARRACH(18) procedure is widely used. Although this procedure provides relief of pain, some problems have been reported such as wrist instability, ulnar deviation of the carpal bones and the rupture of a finger extensor.

Sauve-kapandji(1) procedure is used to treat patients with post traumatic DRUJ problems that include arthritis, instability of DRUJ and Ulna plus variance(3) , or rheumatoid arthritis with destruction of DRUJ(4) It relieves pain, corrects deformity and restores forearm rotation (5) Ulnar carpal shift a known complication of Darrach's procedure is prevented (6) It is shown to provide a stable Ulnar side support to the wrist with DRUJ degeneration (7). The S-K procedure has an undesirable potential of developing an unstable ulnar stump. TALEISNIK (8), reviewed the results of the S-k procedure in 23 patients, who had post traumatic degeneration of DRUJ. He reported that nine patients had pain related to the ulnar stump and two patients needed a repeated operation for instability of ulnar stump. SANDERS et al;(9) in a study of 9 patients who had S-k procedure performed for post traumatic arthritis of DRUJ, reported that two patients had painful instability of the ulnar stump. NAKAMURA et al, (10) performed S-K procedure in 15 patients with chronic dislocation of DRUJ. Functional results were favourable in all patients, homever radiological examination revealed an unstable proximal ulnar stump and radio-ulnar convergence in all patients, and four patients had painful instability of the ulna. MINIAMI et al, (11) reported that S-K procedure gave satisfactory results in 15 patients with arthritis of DRUJ. However 12 patients showed unstable proximal stump and radio-ulnar convergence and 2 patients had persistent pain in the ulnar stump. WAIZENGGER et al, (12) in a series of 14 patients reported four cases of unstable proximal ulna presenting with pain and feeling of insecurity. KAPANDJI (13) recommended that resection should be as distal as possible above the remaining head of the ulna, leaving a short fragment of distal ulna and a smaller ulnar gap of not more than 10mm, to reduce instability of the ulnar stump. The remaining structures that stabilize the ulnar shaft after S-k procedure are the interosseous membrane providing static stabilization and the ECU, FCU tendons and the pronator quadratus providing dynamic stabilization (14) . Release of pronator quadratus from the ulna and reattachment to the ECU in the area of resection helps to prevent ossification and acts as a spacer to prevent impingement between the radius and ulna during forearm rotation (14).

Few procedures to stabilize the ulna have been described. BREEN & JUPITER (15) described a procedure using the ECU and FCU. The tenodesis using a weave of a distally based slip of FCU and a proximally based slip of ECU to stabilize the distal ulna after a Darrrach procedure. ROOTHWELL et al, (16) described a procedure of stabilizing the ulna shaft by suturing the sheath of the ECU and Periosteum as a single layer firmly over the ulnar stump and pseudoarthrosis. LOW et al, (17) described a procedure of stabilizing the ulna, by creating a sling using the radial half slip of ECU tendon and looping it around the ulnar half of the ECU to stabilize the proximal ulnar stump and prevent radial or ulnar deviation and dorsal or volar displacement of bone. TAKUYA et al, (19) used distally based slip of ECU to stabilize the proximal ulnar stump with 80% excellent results with no patient having pain over the ulnar stump.

In our study,a sling was created using the ulnar half slip of ECU tendon based proximally that was looped around the proximal ulnar stump and sutured back to the tendon (ulnar half) of ECU. It was effective as no pain or feeling of instability of the ulna shaft was reported.

Conclusion:

Our modified Sauve-Kapandji procedure is a simple technique for the treatment of DRUJ derangements that makes it possible to stabilize the proximal ulnar stump without limiting the forearm rotation, relieving the pain and maintaining the stability of the wrist.

Refrences:

1. Sauve L;Kapandji M: Nouvelle technique de traitment chirurgical des luxations recidivantes isoleas de l'extremite imferieure du cubitus. J.Chirurgie 1936;47:589-94.
2. Cooney WP; Bussey R; Dobyns JH; Linscheid RL: difficult wrist fractures.Perlunate Fracture dislocations of the wrist: Clin.orthop 1987;214:136-47.
3. Gordon L; Levinsohn DG; Moore SV; Dodds RJ; Castleman LD: The sauve-kapandji procedure for treatment of post traumatic distal radio ulnar joint problems. Hand clin1.1991;7:397-403.
4. Milroy P; Coleman S; Ivers R: The sauve kapandji operation.Technique and results. J Hand surgery 1992;17B:411-4.
5. Mikkelson SS; Lindblad BE; Larsen ER; Sommer J: Sauve kapandji operation for disorders of the distal radio ulnar joint after colle's fracture. Acta orthop.Scand 1997;68:64—6.
6. Goncalves D; De Janeiro R: correction of disorders of the DRUJ by artificial pseudoarthrosis of the ulna. JBJS 1974;56B:462-4.
7. Vincent KA; Szabo RM; Agee JM: The S-K procedure for reconstruction of the rheumatoid DRUJ. J Hand Surg 1993;18-A:978-83.
8. Taleisink J: The S-K procedure. Clin.orthop 1992;275:110-24.
9. Sanders RA; Fredrick HA; Hontas RB: The S-K procedure. A salvage operation for the DRUJ. J Hand.Surg 1991; 16A:1125-9.
10. Nakamura R; Tsunoda K; Watanbe K; Horii E; Miura T: The S-K procedure for chronic dislocation of the DRUJ with destruction of the articular surface. J Hand Surg.1992;17B:127-32.
11. Minami A; Suzuki K; Suenaga N; ishikawamJ: The S-K procedure for osteoarthritis of the distal radioulnar joint. J Hand Surg.1995;20A:602-8.
12. Waizenegger M, Schranz P; Barton NJ: the kapandji procedure for post traumatic problems. Injury 1993,24:662-6.
13. Kapandji AL: The sauve kapandji procedure. J Hand Surg.1992;17B:125-6.
14. Lamley DM; Fernandez DL: Results of the modified S-K procedure in the treatment of chronic post traumatic derangement of the distal radioulnar joint.JBJS 1998;1758-69.
15. Breen TF; Jupiter JB: Extensor carpi ulnaris and flexor carpi ulnaris tenodesis of the unstable distal ulna. J Hand Surg.1989;14A:612-7.
16. Rothwell AG; O'Neil L; Cragg K: The S-K procedure for disorders of the DRUJ.A simplified technique. J Hand Surg.1996;21A:771-7.
17. Low CK; Chew WYC: Results of Sauve Kapandji procedure. Singapore med.J.2002 Vol43(3);137.
18. Darrach W: Anterior dislocation of the head of ulna. Ann.Surg.1912:56:812.
19. Takuya S; Yoshita N; Yasumasa S; Gembun Y; Hiroshi H; Tomoyuki R: A suspencion procedure using ECU tendon for DRUJ disorders. J Nippon Med Sch 2001;68(3).

INCIDENCE OF MACULOPATHY IN NON-PROLIFERATIVE AND PROLIFERATIVE DIABETIC RETINOPATHY

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ABSTRACT: *Diabetic retinopathy, a progressive long term complication of diabetes mellitus is one of the commonest causes of blindness world over. The disease is responsible for blindness either in its proliferative form (proliferative diabetic retinopathy) or due to diabetic maculopathy selectively affecting the macular area. Diabetic maculopathy may be associated with changes of non-proliferative or proliferative diabetic retinopathy.*

A total of 200 eyes of 100 subjects were examined Group I comprised of 100 eyes of 50 patients having diabetes of varying duration and having changes of non-proliferative diabetic retinopathy and Group II comprised of 100 eyes of 50 patients having diabetes of varying duration and changes of proliferative diabetic retinopathy. Incidence of maculopathy was studied. 42% of patients in Group I and 70% of patients in Group II were found to have maculopathy. The presence of diabetic maculopathy was seen to be directly related to the overall severity of the systemic disease as well as to the duration of diabetes.

Key word : *Non-proliferative diabetic retinopathy, proliferative diabetic retinopathy, diabetic maculopathy, macular edema.*

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MATERIALS AND METHODS

The present study was carried out on patients attending the outpatients department of the Department of Ophthalmology, SMHS Hospital, Srinagar from April 1998 to July 2000.

The study included 200 eyes of 100 patients, Grouped as below:-

GROUP I : included 100 eyes of 50 patients having non-proliferative diabetic retinopathy of varying duration. Further, Group I was subdivided in Group Ia (patients without maculopathy) and Group Ib (patients with maculopathy).

GROUP II: included 100 eyes of 50 patients having proliferative diabetic retinopathy of varying duration. Group II was further sub-divided into Group IIa (patients without maculopathy) and Group IIb (patients with maculopathy).

The Fundus examination of each patient besides routine direct ophthalmoscopy included indirect ophthalmoscopy, slit lamp bio-microscopy and fundus fluorescein angiography. Special emphasis was given to the presence of microaneurysms, cottonwool spots, hard exudates, dot and blot hemorrhages, lipid deposits, macular edema, presence of shunt vessels, neo-vascularization, subhyloid or frank vitreous hemorrhage, fibrovascular proliferation and tractional retinal detachment.

RESULTS

In Group I, 21 patients out of 50 (42%) subjects had diabetic maculopathy, an incidence of 420 per thousand while in Group II, diabetic maculopathy was observed in 35 patients out of 50 (70%) subjects, an incidence of 700 per thousand.

In Group I, females (56%) were affected more frequently than males (44%) while in Group II, males (58%) were affected more frequently than females (42%). **[TABLE 1]**

TABLE – 1: **Showing age and Sex distribution of subjects in different Groups (Group I and Group II)**

Age Group in years		*Group I*			*Group II*	
	Male	*Female*	*Total*	*Male*	*Female*	*Total*
<30	2	1	3	7	2	9
41–50	11	10	21	13	9	22
51–75	9	17	26	9	10	19
Total	22	28	50	29	21	50

$X^2$1.df=1.527 P:NS $X^2$1.df=0.891 P:NS

NS: Not-significant Statistically

The age of the patients of non-proliferative diabetic retinopathy with maculopathy (Group Ib) ranged from

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18-60 years with an average of 49 years while the patients of proliferative diabetic retinopathy with maculopathy (Group IIb) had an average age of 47 years with a range from 16-65 years **[TABLE 2]**

TABLE – 2: Showing age and sex distribution of subjects in diabetic maculopathy Groups (Group Ib and Group IIb)

Age Group in years	Group I b Male	Female	Total	Group II b Male	Female	Total
<30	2		2	1	2	3
31–50	3	5	8	10	6	16
> 51	4	7	11	7	9	16
Total	9	12	21	18	17	35

In Group I, 62% had a duration of diabetes ranging between 6-15 years. The average duration of diabetes for this Group was 10.3 years [TABLE 3]. Patients who had associated maculopathy (Group Ib) had an average duration of diabetes greater than seen in patients without maculopathy (Group Ia) [TABLE 4]

TABLE – 3: Showing duration diabetes in Group I and II

No. of years	Group I No. of subjects	Percentage	Group II No. of subjects	Percentage
0-5 years	11	22	2	4
6-15 years	31	62	44	88
> 16 years	8	16	4	8

X^2 1.df=9.814 ** P value <.01

** Significant at 1% level of significance.

TABLE – 4: Showing average duration diabetes in Sub-Groups

Group	No. of Patients	Average duration of diabetes in years
Group Ia	29	9.5
Group Ib	21	14.4
Group IIa	15	8.6
Group IIb	35	12.2

In Group II, 88% had a duration of diabetes ranging between 6-15 years. The average duration was 11.1 years **[TABLE 3]**. Patients of this Group with maculopathy (Group IIb) had an average duration of diabetes greater than seen in subjects without maculopathy (Group IIa) **[TABLE 4]**.

In Group I, 68% were controlled on various oral hypoglycemic agents while 32% patients required insulin. In patients with maculopathy (Group Ib) 76.19% were controlled on various oral hypoglycemic agents while 23.81% patients required insulin. In Group II, 42% and 58% of patients were controlled on oral hypoglycemic agents and insulin respectively. In patients with maculopathy (Group IIb) the figures were 45.71% and 54.29% respectively **[TABLE 5]**.

TABLE – 5: Showing different forms of antidiabetic treatment in different Groups

Group	No. of cases controlled on diet alone	No. of cases on oral hypoglycemic agents	No. of cases total on insulin	Total
Group I	-	34(68%)	16(32%)	50
Group Ia	-	18(62.06%)	11(37.94%)	29
Group Ib	-	16(76.19%)	5(23.81%)	21
Group II	-	21(42%)	29(58%)	50
Group IIa	-	5(33.33%)	10(66.67%)	15
Group IIb	-	16(45.71%)	19(54.29%)	35

*X^2 1.df=6.826 ** P Value <0.01*

*** Significant at 1% level of significance.*

DISCUSSION

Diabetic retinopathy may cause blindness in both the proliferative and background stages of the disease. In the latter, the visual impairment is caused by a direct involvement of the macular area, a condition to which the term maculopathy is often applied and is seen more frequently among older onset type II diabetes.

Diabetic maculopathy in contrast to proliferative diabetic retinopathy is a silent killer as the macular changes being invariably bilateral and symmetrical lead to progressive deterioration of vision over a long period of time. The most important component of diabetic maculopathy is the edema of the macular area which varies from mild to severe often with a marked exudative component. The edema is its early stages is often reversible. On the other hand, when it is associated with micro and macrocystic changes, the condition is often irreversible, although stabilization and lack of progression can take place in some instances with diabetic control and laser treatment. Visual loss due to diabetic maculopathy is 72% in blind and partially sighted diabetics [1].

In the present study 56 out of 100 (56%) patients were found to have diabetic maculopathy. This condition is seen in association with either non-proliferative or proliferative diabetic retinopathy. 42% of patients were found to have maculopathy associated with non-proliferative diabetic retinopathy and 70% of patients had maculopathy associated with proliferative diabetic retinopathy.

Our findings suggest that diabetic maculopathy appears more frequently with proliferative than non-proliferative diabetic retinopathy. Aiello et al [2] (1982) in their study observed that maculopathy occurred at all stages of retinopathy. Bodansky et al[3] found 43% of cases having maculopathy in a study of 100 consecutive diabetic patients. Clark et al [1] (1994) reported an incidence of 72% of diabetic maculopathy in his series. The incidence of macular edema in patients with mild to severe non-proliferative and moderate to high risk proliferative diabetic retinopathy was 81.6% in a series reported by Nizan Kowska et al [4] (1995). Our results resemble the work of

Kleen R et al [5], 1984 who reported an incidence of 38% of macular edema among eyes with moderate to severe non-proliferative diabetic retinopathy and 71% among eyes with proliferative diabetic retinopathy.

Our findings regarding sex suggest that females were affected more frequently in Group I while males were affected more frequently than females in Group II. The overall male : female ratio in the two maculopathy Groups (Group Ib and IIb) was 27:29 showing a slight predominance of females in patients with diabetic maculopathy. However, statistically the difference between the two sexes was insignificant in all Groups. Our observations regarding age suggest that diabetic maculopathy occurs more frequently in older onset diabetics. Among the juvenile onset diabetics, proliferative diabetic retinopathy was seen more frequently. Statistically there was a significant difference ($P<.001$) in age among different Groups.

The duratibn of diabetes is considered as the most important predictor for the development of diabetic retinopathy [El. Hifnawy et al [6] (1992), Ballard et al [7] (1986)]. In our study we observed an average duration of diabetes of 10.3 years in patients with non-proliferative diabetic retinopathy (Group I) and 11.1 years in patients with proliferative diabetic retinopathy (Group II). Patients with associated maculopathy in both Groups (Group Ib and IIb) had an average duration of diabetes [14.4 and 12.2 years respectively] greater than seen in patients without maculopathy [Group Ia and IIa, 9.5 and 8.6 years respectively]. These observations reflect an increasing risk of developing diabetic retinopathy, particularly proliferative diabetic retinopathy as well as diabetic maculopathy with increasing duration of diabetes. Statistically, there was a significant difference ($P <.01$) in the duration of diabetes among various Groups. Patz and Murphy[8] (1985), observed diabetic retinopathy in 50% of patients with duration of diabetes of 10 years, in 70% with duration of 15 years and in 95% of the patients with duration of 25 years. In Pima Indians, the incidence of proliferative diabetic retinopathy was strongly related to the duration of diabetes (Nelson et al [9] 1989).

Our observations regarding the type of diabetes and antidiabetic treatment suggest that non-proliferative diabetic retinopathy was seen more frequently in NIDDM patients (Group I), while proliferative diabetic retinopathy was seen more frequently in insulin users (Group II). There was a significant difference statistically ($P <.01$) among patients on oral hypoglycemic agents and insulin users. Grey et al[10] (1986), noticed a higher prevalence of both background and serious retinopathy in IDDM and NIDDM patients. Insulin users were a relatively high risk Group for the development of proliferative diabetic retinopathy in the older onset Group in WESDR[11] (1989). In the Pima Indians, proliferative diabetic retinopathy was associated with insulin therapy (Nelson et al[9] 1989). Similar observations were made in South Africa. (Mouton et al[12], 1988). Our observations suggest that diabetic maculopathy was predominantly seen in non-insulin dependent type II diabetes mellitus [32 out of 56 subjects (57.14%)] than insulin dependent type I diabetes mellitus. [24 out of 56 subjects (43.85%)]. However, as an individual Group, maculopathy was seen more frequently in insulin users in Group IIb i.e; patients with proliferative diabetic retinopathy. These observations can be explained on the fact that proliferative diabetic retinopathy was seen more frequently in insulin users in this study as already explained. Aiello et al[2] (1981), reported that maculopathy is much more common in older people with type II diabetes than in those with type I disease and as such is related to the age of the person affected. They also noted that when young persons with type I diabetes had maculopathy, they usually also had proliferative retinopathy.

Diabetic maculopathy because of its varied clinical manifestations presents differently in different patients. A good number of patients with early diabetic retinopathy are usually asymptomatic. These are usually the cases who have diabetes of lesser duration and are probably having a reasonable control of diabetes. It is now generally agreed that fundus changes in a diabetic are usually not clinically apparent before five years of systemic disease. This observation assumes special significance in relation to the development of diabetic maculopathy. With the development of edema, exudates, microaneurysms and hemorrhages in the macular area which is related to the duration of diabetes and improper diabetic control, the patient starts complaining of visual symptoms and dysfunction.

Although depending upon the predominant clinical features, the maculopathy was subdivided into three clinical types; focal (exudative, fig. 1), diffuse (edematous, fig. 2) and ischemic variety (fig. 3). There is often an overlap between each clinical entity. Many reports do not distinguish among the various forms of diabetic maculopathy, but only discuss the macular edema which is the first and dominant sign of maculopathy[13].

TABLE – 6: Showing different types of diabetic maculopathy

Types of diabetic maculopathy	Group I b	Group II b		
	No. of eye	Percentage	No. of eyes	Percentage
a) Focal	20	62.50	13	26
b) Diffuse	7	21.87	20	40
c) Ischemic	5	15.62	17	34
Total	32	100	50	100

*X^2 2.df=10.851** P value <0.01*

*** Significant at 1% level of significance*

In the present study, focal (exudative) maculopathy

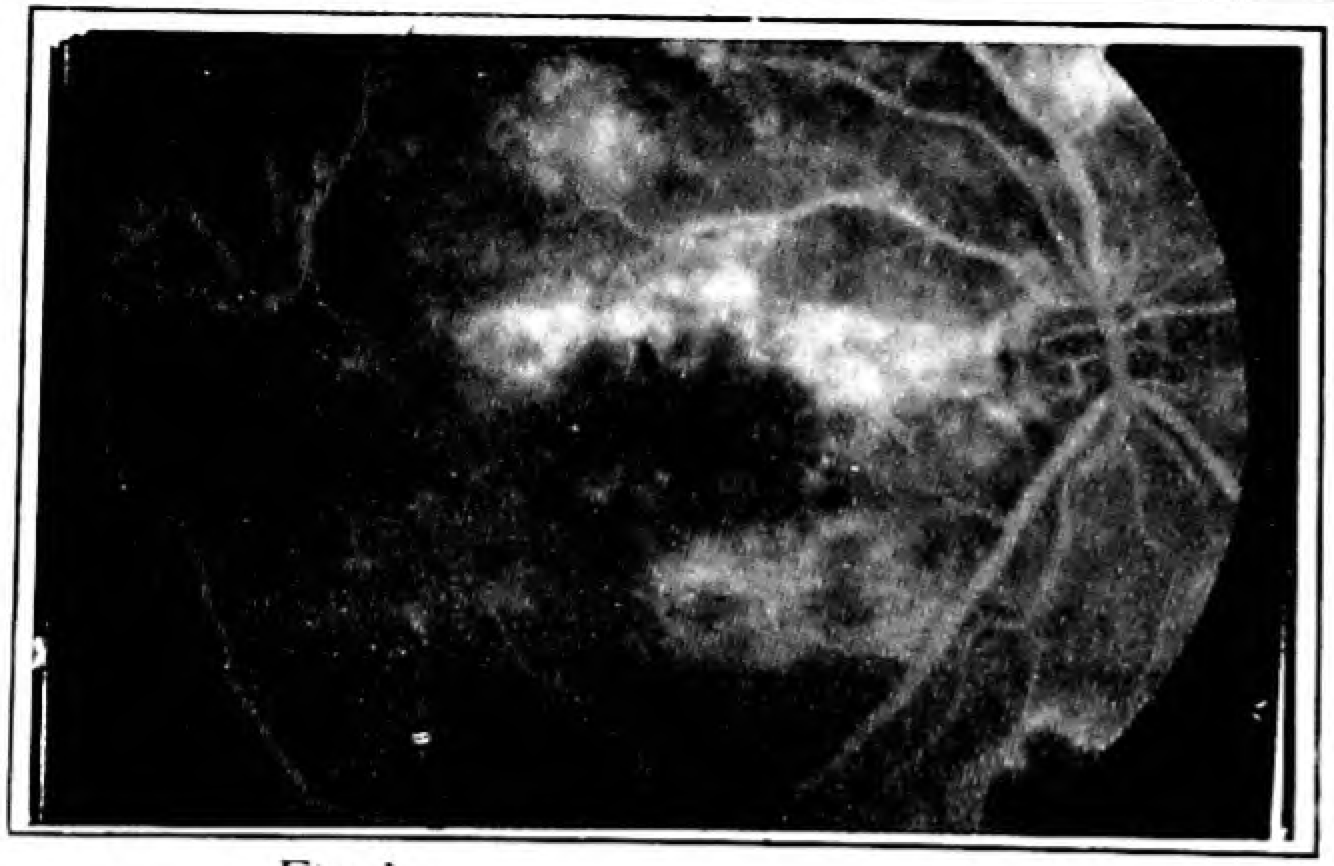

Fig. 1

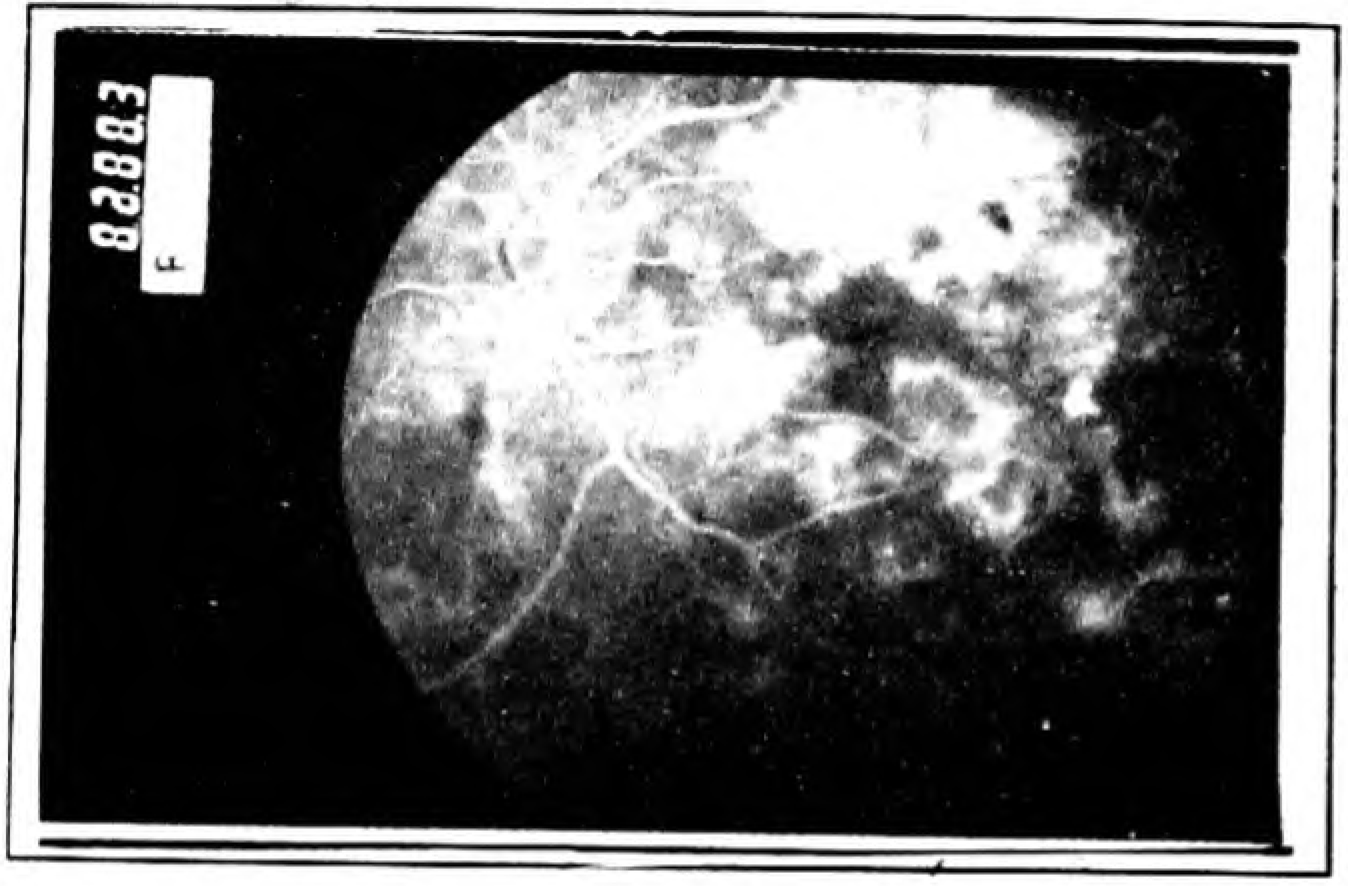

Fig. 2

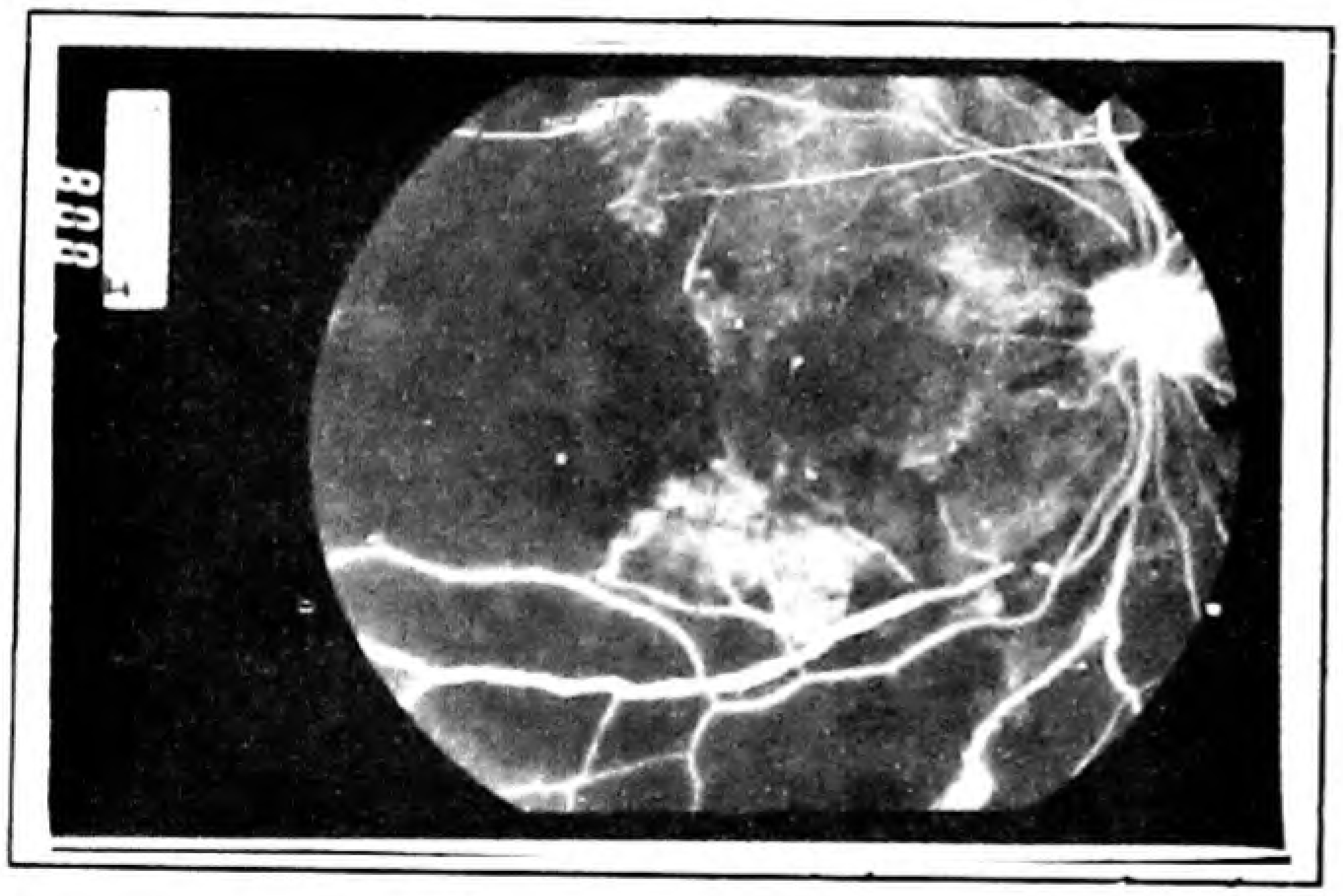

Fig. 3

was the commonest clinical type seen in Group Ib (62.50%) while diffuse maculopathy (edematous) was the commonest type seen in Group IIb (40%), Ischemic maculopathy was seen in 15.62% and 34% of eyes in the two Groups respectively and was more common in eyes with proliferative diabetic retinopathy [P<0.01 **(TABLE 6)**].

Although diabetic maculopathy has been classified into different entities for the sake of natural course of each variety, [Siegelman[14] (1979), Whitelock, Kearns, Blach[15] (1979)] the clinical manifestation of the diabetic process was not confined to the macular area alone. The maculopathy is invariably associated with other changes of either proliferative or proliferative diabetic retinopathy (Kohner[16] 1978).

REFERENCES:

1. Clark JB; Grey RH; Lim KK; Burns-Cox CJ : Loss of vision before ophthalmic referral in blind and partially sighted diabetics in Bristol.Br J Ophthalmol. 1994, Oct. 78(10); 741-4.
2. Aiello LM; Rand LI; Briones JC; Wafai MZ; Sebestyen JG; Diabetic retinopathy in Joslin Clinic patients with adult onset diabetes. Ophthalmology 1981; 88; 619-623
3. Bodansky HJ; Diabetic retinopathy and its relation to type of diabetes. Br J Ophthalmol 1982; 66: 496-499.
4. Nizan Kowska MH; Incidence of and treatment outcome for diabetic maculopathy. Klin Oczna 1995; 97: 282-5.
5. Klein R; Klein BEK; Moss SE; et al. The Wisconsin Epidemiologic study of Diabetic Retinopathy (WESDR) IV. Diabetic macular edema. Ophthalmology 1984; 91; 1404-1474.
6. EL-Hifnawy MAM; Fadel AM; Atiea JA: Risk factors for diabetic retinopathy. Saudi J. Ophthalmology 1992; 6(1): 3-9.
7. Ballard DJ; Melton LJ; Dwyer MS; et al. Risk factors for diabetic retinopathy. Diabetes care, 1986; 9(4) 344-42.
8. Patz A; Murphy RP; Diabetic retinopathy. In Diabetic Retinopathy 1985, held at Wilmer Ophthalmological Institute, the Johns Hopkin's Hospital, Baltimore, MD, USA. Nov 1, 1985.
9. Nelson RG; Wolfe JA; Horton MB; et al. Proliferative diabetic retinopathy in NIDDM. Incidence and risk factors in Pima Indian's. Diabetes, 1989; 28(4): 435-40.
10. Grey RHB; Malcolm N; Orcilly D; et al. Ophthalmic survey of a diabetic clinic. Br J Ophthalmol 1986; 70 : 797-803.
11. Klein R; Klein BEK; Moss SE; et al. The Wisconsin Epidemiologic study of Diabetic Retinopathy (WESDR) X. Four years incidence and progression of diabetic retinopathy when age at diagnosis is 30 years or more. Arch Ophthalmology 1989; 107: 244-9.
12. Mouton DP; Gill AG; Prevelance of diabetic retinopathy and evaluation of risk factors. S Afr Med J, 1988; 74(8): 399-402.
13. Ivanisevic M; Diabetic maculopathy. Lijec. Vjesn. 1992; 114 : 9-12 ; 300-3.
14. Siegelman JL : Diabetic maculopathy, American Academy of Ophthalmology 1979.
15. Whitelock M; Kearns; Blach and Hamilton: The diabetic maculopathies. Trans Ophthal Soc. UK, 1979 : 99 : 314.
16. Kohner EM; The evolution of natural history of diabetic retinopathy. Int. Ophthalmol Clin 1978; 18: 1.

PEAK EXPIRATORY FLOW RATES IN YOUNG KASHMIRI ADULTS THROUGH ELECTRONIC SPIROMETRY

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Abstract: *Peak Expiratory Flow Rate was conducted on young healthy non-smoking permanent residents of Kashmir for both sexes. The subjects were mostly 17-30 years old. The random sample from population constituted mostly students, attendants of patients and medical employees. Five readings were taken and the highest value among them was considered for the study. PEFR values for Kashmiri men were significantly higher than those in women. The values for both the sexes were higher for Indian norms as recorded till date and matched in both the sexes to the western norms [1], while norms are below reference values for the western literature available.*

Key Words: *Electronic Spirometer, Peak Expiratory Flow Rate, B.T.P.S., Regional variation.*

JK-Practitioner 2003;10(4): 279-280

Introduction:

It is well documented in the literature that the wide range of geographical, climatic, anthropometric and socio-economic conditions of India are associated with regional differences in lung functions.

Single breath pulmonary function tests are widely used in the modern computerized scientific age. Expiratory flow reaches a transient peak early in forced expiratory maneuver. Peak flow occurs during the high effort dependent portions of the expiratory maneuver and decreased values can result from even slightly submaximal effort rather than from airway obstruction. Nevertheless, the ease of measuring peak flow with an inexpensive small portable device has made it a popular means of following the pattern of airflow obstruction on an ambulatory basis, as in patients suspected of having occupational disorders and in those who seem insensitive to the severity of bronchospasm, PEFR plays an important role as it is largely a function of large airways. It is also effected by expulsion of air from compressed central airways. For these reasons, peak flow is abnormally decreased only in moderate to severe airway obstruction.

However, different studies have yielded different sets of regression equations[2] as predictors of PEFR for the populations, they have studied with the aim of obtaining data so that they could be used as references.

Since multiple ethnic population with different regional parameters like free air pollution, body size, chest circumference and altitude giving norms in India [3], needed this study to be conducted as quite less work has been reported from northern India with modern computerized equipment. The present study reports the results of PEFR in a group of healthy young non-smoking Kashmiri young adult population.

Material and Methods:

212 healthy non-smoking young adults from Kashmir valley (altitude 5000 ft.) of both sexes, males (126) and females (86) in the age group of 17-30 yrs were included in this study after taking a detailed history and thorough examination fulfilling the criteria laid down by A.T.S (1991)[4]. The procedure was demonstrated by a skilled person to subjects before recording, which was done same time every day from May to June as there is circadian rhythm where for PEFR the peak to trough amplitude is in order of 8%. (A.T.S) (1991)[4].

The subjects were motivated during the maneuver, which was done using MEDISPIROR-ELECTRONIC SPIROMETER in standing position. The electro-mechanical pneumatach was put inside the mouth after sterilizing it's mouthpiece. The procedure was recorded for 10 seconds. The flow and volume output of PEFR maneuver accepted for the study was confirmed by Flow Vol. plot (graph) and Vol./Time plot (graph) in accordance with above recommendation.

The Spirogram, where subject exhibited maximum efforts with highest values on Vol./Time plot were taken for study. The printed values and graphs were instantly produced and corrected to B.T.P.S by electronic apparatus itself.

Results:

The physical characteristics of various parameters in the study are given below alongwith PEFR values.

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1.	AGE	Mean	Males	20.9 yrs S.D ± 1.25
			Females	20.45 S.D ± 1.001
2.	HEIGHT	Mean	Males	172.24 cms. S.D ± 6.537
			Females	159.42 cms. S.D ± 5.292
3.	CHEST/EXPANSION	Mean	Males	Inspiration 90.952cms. S.D.± 7.096 Expiration 83.055 cms.S.D. ±6.875
			Females	Inspiration 87.09cms. S.D.± 6.3996 Expiration 80.54 cms.S.D. ±7.103
4.	PEFR	Mean	Males	9.62195 S.D. ± 1.7435
			Females	6.78361 S.D. ± 1.32482

Discussion:

Pulmonary function tests are important in the present scenario of computer age in clinical medicine and the values are influenced by race, age, sex, height and weight, altitude, temperature and lately air pollution, passive smoking. automobile exhaustion, false values for legal battles over fitness, not to speak of technical experience and instrument standardization. Expiratory flow rates have lately gained considerable importance as they have been found to help detect airway obstruction in different segments of broncho-pulmonary tree. It is an indication of status of more distant airways as they are dependant on voluntary effort and are reproducible. The airways less than 2 mm in diameter are the sites were obstruction processes first begin and which are not picked up by conventional spirometric tests (Macklen 1971 A.D.) [5].

In the present study the values of PEFR in case of both males as well females showed a downward trend with respect to age and sex and a positive relationship with respect to height. These values match most of the western literature closely. From the Indian literature available, Mahajan [6] (1997 A.D.)in his study showed higher values of 6.3 lit. in comparison to slightly lower values of 5.3 lit. from another study by Gupta (1975 A.D.)[7].The values in our study were, however, closer to those shown by Peter (1967)[8] in his study. Even James. K. Stroller (1993 A.D)[9] in a study carried out by him showed a value of 8.84 lit., which is also lower than that of our's. Till now in India Prateeb K.Mehrotra (1997) in a study in Lucknow showed values of 6.9±1.8.lit.

The reason for getting higher values in the present study, could be attributed to the fact that Kashmir is at a higher altitude. Altitudes higher than 1500 m do appear to cause measurable changes in lung volumes and flow rates which may be affected by changes in air density as FEV are slightly increased s because of decrease in air density. Also those residing probably have large lung volumes than do at low altitude as because of hypoxia the accessory muscles of respiration are far more developed, this difference in muscle effectiveness with lung-volume may reflect difference in length-tension/or geometric mechanical advantage between the rib cage and diaphragm, further the size of lung may also be effected by increase in physical activity of high-landers thus effecting the flow rates and lastly due to anthropometric ratios the high- landers have a high chest to leg ratio thereby increasing the vertical size of the chest hence the volumes.(A.T.S. 1994).[10] At high altitude detrimental effect is hypoxia. (Woolcock 1972) [11,12,13] and also of better sanitary condition and less population congestion with modern instruments[14] here as compared to rest of India where there is poor sanitary conditions, population over-growth and repeated infections, deficiency diseases that effect the lung growth physiology. The higher values, and clinical importance of this place due to the higher prevalence of COPD especially the asthmatics makes a need for a broader study necessary in this part of India as most of the expert panel reports in western countries heavily depend on informed use of peak flow meters for the proper patient care.[14]

References:

1. Natrajan S.Rahha. PEFR in normal south Indians.Ind.Journal of chest diseases and allied sciences.1978:20;178-182.
2. Singh. H.D,Sudesh.PEFR in south Indian adults. Ind. Journal of Physiology and Pharmacology. 1979: 23;315-20.
3. Jain S.K and Gupta.Age,height and B.S.A as determinants of ventilatory norm in healthy males above 40 years age.Ind.Journal of medical research 1967:55;612-19
4. American Thoracic Society. Medical Section of American lung Association. Am. Rev. Resp. Dis. (1991) 144. 1202 - 1218.
5. Macklen P.T: Thmlbeck W.N. (1971) "Chronic obstructive disease of small airways" Amn: Int. Medicine. 76 : 167.
6. Mahajan K.K, Mahajan A. and Mishra (1997).Pulmonary functions in healthy females of Haryana. Ind .Journal of chest allied Sci. 1997: 39;163-171.
7. Gupta S; M:B. Puri (1975) Pulmonary function test in health Jr. Assoc.: Physiology Ind: 23 247-252.
8. Peters J.M. & B.G. Ferrir Jr. Pul: Function and Respiratory system in College age group Am. Rev. Resp. Dis. 95 774 - 782; 1967.
9. James.K.Stroller. Basueda.S. (1993) "Trial of standard versus modified expiration to achieve end of spirometry criteria Amn. Rev: Resp: Dis. (1993) Vol: 148 275 - 280.
10. A.T.S. of American Lung Association "Standardization of spirometery (1994) update" Am. J.Resp. Crit Care Med. 1994 : 152: 1107-1136.
11. N Woolcock A.J,Colman M.H and Blackburn C.R.B.Factors affecting normal values for ventilatory lung functions. Am. Rev. Respir. Dis.1972:106;692-708.
12. A.Brancatisano. L.A. Engel. S.H.Lorins Lung vol. & effectiveness of inspiratory muscles. Jr. of applied Physiology (1993) Feb: No. 2, vol: 74. 688 - 694.
13. K.K.Mahajan: Pulmonary function and their correlation with anthropomorphic parameters in young adults of Haryana Ind: J. Physiology. Pharm: Jan - March (1978); page 88 - 92
14. Linn W.S and Soloman J.G. Standardization of multiple spirometers at widely separated time and place. Am.Jr.Resp.Crit Care Med.1996:153(4);1309-13.

INFLUENCE OF CIPROFLOXACIN ON PHAGOCYTIC CAPACITY AGAINST STAPHILOCOCCUS AUREUS

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Abstract: *Ciprofloxacin – is an antimicrobial agent from the class of systemic fluoroquinolones is active against gram-negative bacteria and, from among gram-positive bacteria, it is active especiallly against staphylococci. The studies were undertaken on staphylococci species showing positive characteristics like coagulase, fibrinolysin, haemolysin and concluded that ciprofloxacin is an antistaphylococcal agent which both inhibits the bacterial DNA and has a nonspecific immunostimulant effect.*

Key words: *ciprofloxacin, gram-positive bacteria, coagulase, fibrinolysin, haemolysin*

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Introduction

Ciprofloxacin is an antimicrobial bactericidal agent of the class of systemic fluoroquinolones. Its chemical structure and mechanism of action are shown in fig. 1.

The data reported in the specialized literature demonstrates that fluoroquinolones inhibit the activity of DNA gyrase, an enzyme that takes part in DNA transcription and replication. Thus, it inhibits both DNA synthesis and protein synthesis. The bactericidal activity of fluoroqinolones depends only to a very small extent on dosage[3] and their selective action is due to their interaction with the similar enzymes in the eukariote cells at concentrations 100 – 1000 times higher than the therapeutic concentrations [4, 5]

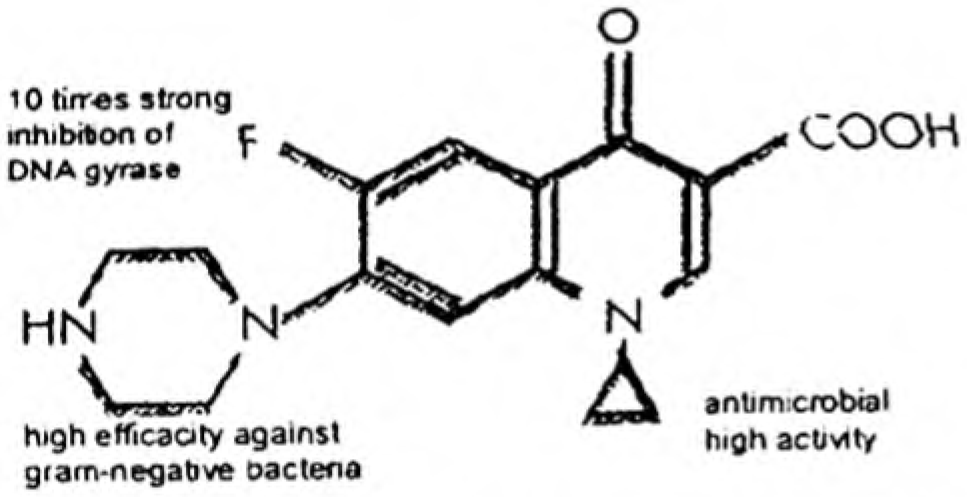
10 times strong inhibition of DNA gyrase
F
O
COOH
HN
N
N
high efficacity against gram-negative bacteria
antimicrobial high activity

Fig. 1. Correlation between ciprofloxacin chemical structure and its action (Chu DTW, Fernandes PB, 1989; Domagala J. M., 1994)[1,2]

The literature reports ciprofloxacin to be active against gram – negative bacteria: Escherichia, Klebsiella, Enterobacter, Citrobacter, Edwardsiella, Salmonella, Shigella, Proteus, Providencia, Morganella, Serratia, Yersinia, Pseudomonas, Acinetobacter, Neisseria, Haemophilus, Campilobacter, Aeromonas, Vibrio, Brucella, Pasteurella, Legionella, and from among gram – positive bacteria, it is active especially against staphylococci [6, 7]

Some studies have shown that intraperitoneal therapy of peritonitis with ciprofloxacin through ambulatory continuing peritoneal dialysis is 75 – 80 % effective, more effective than the oral therapy with the same drug[7, 8]

The mechanism of ciprofloxacin action is not fully known yet, but the studies have indicated that the inhibition of DNA gyrase is not the only mechanism in action[3, 6, 7] This paper investigates whether ciprofloxacin has a nonspecific immunostimulant effect besides the inhibitory effect of the bacterial DNA synthesis or not. For this purpose, we studied the influence of ciprofloxacin on the phagocytic activity of mice leucocytes in vivo and in vitro.

Material and Methods

The studies were made on staphylococci species showing positive pathological characteristics: coagulase, fibrinolysin, haemolysin. The investigation was carried out on 24 hours staphylococci cultures, on blood-agar emulsified in saline solution, with a concentration of 2 million germs/mm^3 according to the nephelometric scale. As bactericidal agent we used Ciprinol in 10 ml vials with a concentration of 100 mg / 10 ml. The dose was 0.5 ml (5 mg) according to the instructions recommended in the literature[7, 8, 9]

I. The phagocytic parameters, Phagocytary Power (Ph P) and Opsono – Cytophagyc Index (O C I) were

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studied in vitro on Batches of 10 subjects, with the classical method. Ciprofloxacin was not added to bath 0.

- Intraperitoneal inoculation of sterile medium
- Taking peritoneal leucocytar sample every 3 hours (0.5 ml)
- Setting it in contact with the microbial suspension (5 ml)
- Addition of ciprofloxacin (5 mg)
- Smearing, smears coloured in methylene blue.

II. For the in vivo study of the phagocytic parameters 10 batches of ten subjects each were used with the classical method:

- Batch 0 – inoculation of sterile medium, followed by the inoculation of the microbial suspension after 3 hours
- Batch 1 – the steps for batch 0 followed by the inoculation of the culture and the inoculation of ciprofloxacin after one hour
- Batch 2 - the steps for batch 0 followed by the concomitant inoculation of the culture and of ciprofloxacin

III. The postantibiotic effect was also studied by analyzing the macrophage response in vivo 12 and 24 hours after administration. This parameter was also studied by comparing batch 0 (without administering ciprofloxacin) with the batches to which the antibiotic had been administered.

Results

I. The in vivo analysis of the media of phagocytic parameters, studied dynamically, led to the results shown in table I and graphs 1 and 2.

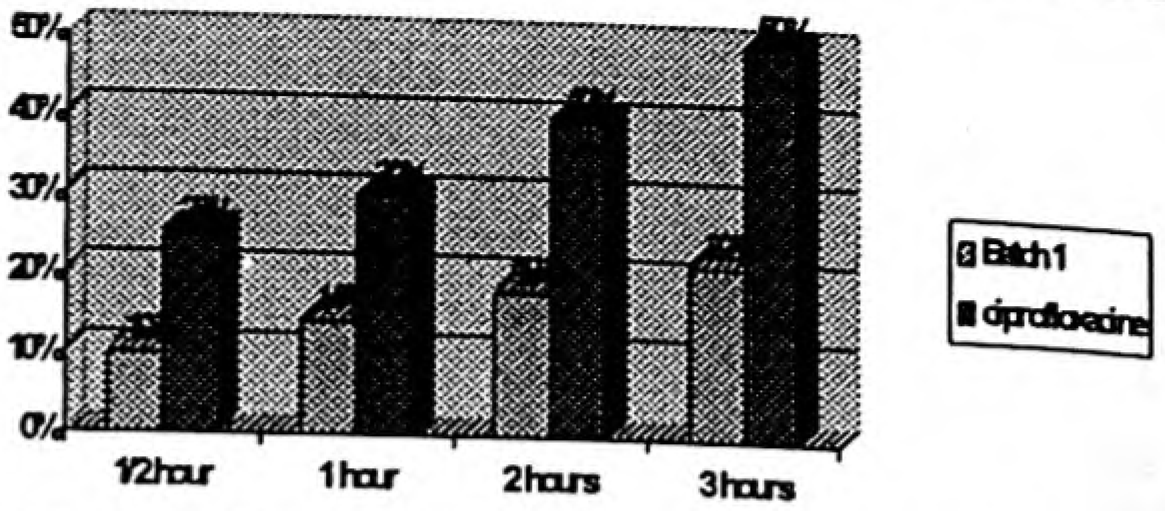
Table I. **The in vivo analysis of the media of phagocitic parameters**

	½ hour		1 hour		2 hours		3 hours	
	Ph P	O C I	Ph P	O C I	Ph P	O C I	Ph P	O C I
Batch 0	10%	1	14%	2	18%	2.5	22%	2.8
Ciprofloxacin	25%	1.8	30%	2.8	40%	3.8	50%	4.5

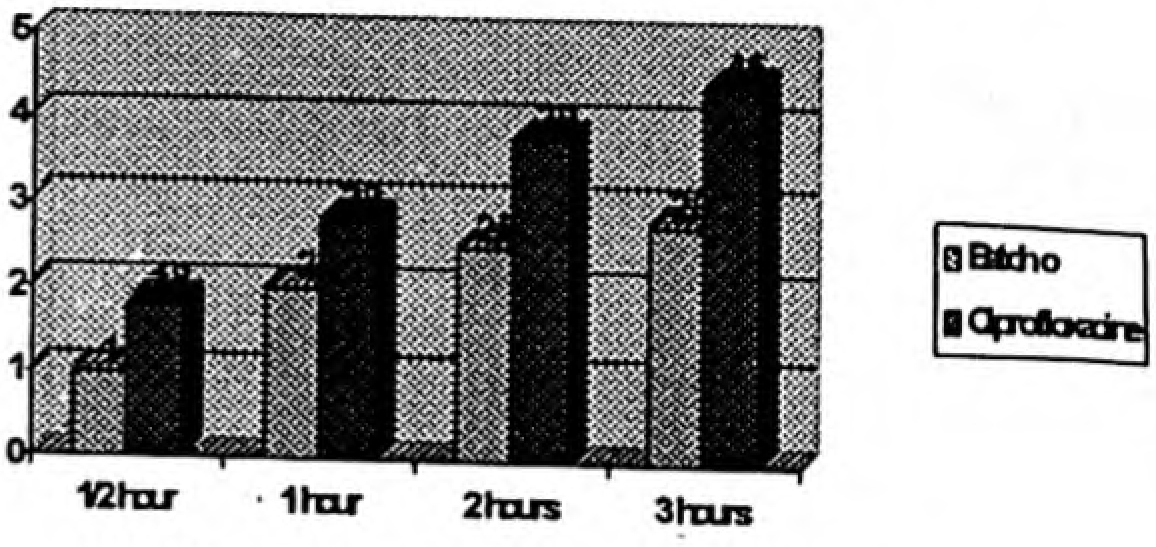
II. The phagocytic response in vivo, presented diagrammatically, is shown in table II and graphs 3 and 4.

Table II. **The phagocytic response in vivo**

	½ hour		1 hour		2 hours		3 hours	
	Ph P	O C I	Ph P	O C I	Ph P	O C I	Ph P	O C I
Batch 0	20%	2	30%	2.8	40%	3.5	45%	4
Batch 1	30%	2.3	38%	3	42%	3.6	58%	5.3
Batch 2	31%	2.3	40%	3	42%	3.8	65%	5.5



Graph 1. Comparative evolution of Ph P in vitro

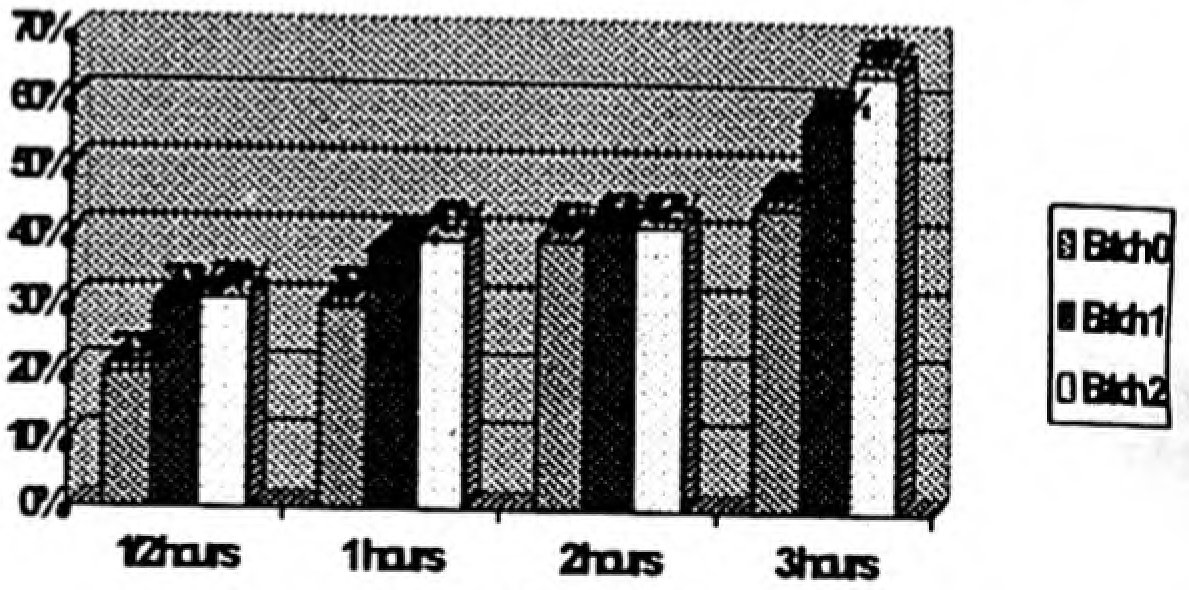


Graph 2. In vitro O C I evolution

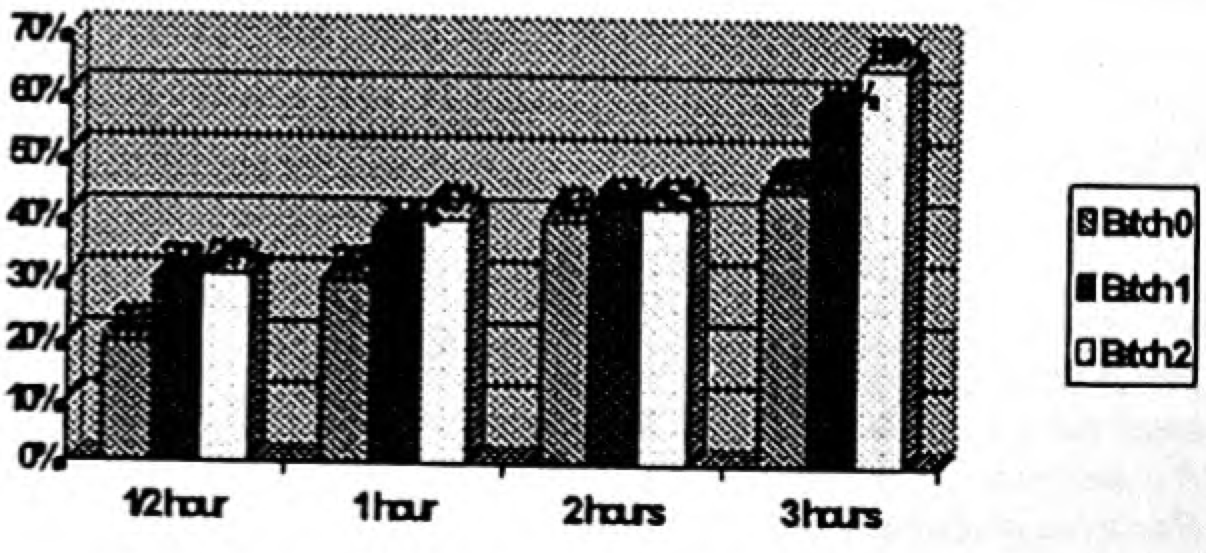
III. The in vivo macrophage response studied at 12 and 24 hours has yielded the results exposed in table III and graphs 5 and 6 in details.

Table III. **The in vivo macrophage response studied at 12 and 24 hours**

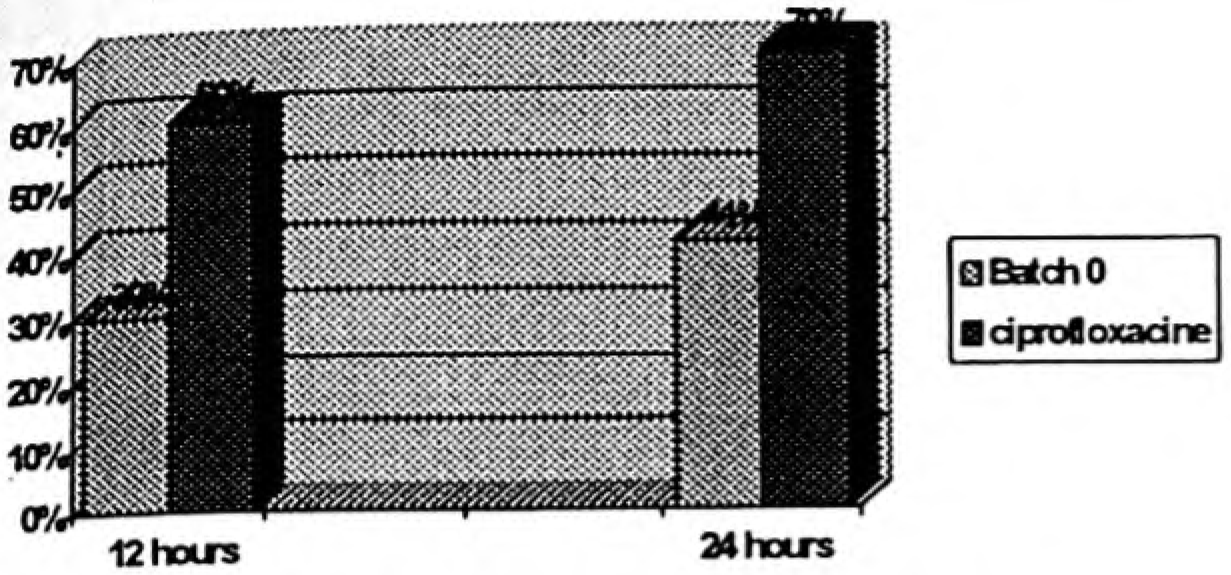
	12 hours		24 hours	
	Ph P	O C I	Ph P	O C I
Batch 0	30%	3.1	41%	3.8
Ciprofloxacin	60%	5	70%	6



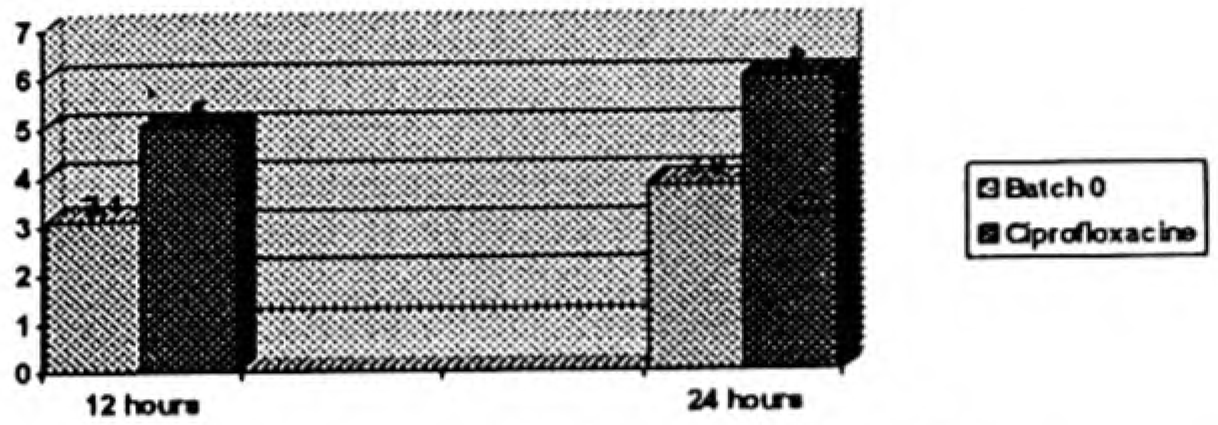
Graph 3. In vitro Ph P evolution



Graph 4. In vivo O C I evolution



Graph 5. Late evolution of Ph P in vivo



Graph 6. Late evolution of O C I in vivo

Conclusions

- ◆ The analysis of the results enables us to conclude that ciprofloxacin is an antistaphylococcal agent, which both inhibits the bacterial DNA and has nonspecific immunostimulant effect due to the increase of phagocytic activity of the macrophage and of the neutrophils.
- ◆ The phagocytic activity persists intensely even at 12 – 24 hours after administration, although the turnover of Ciprinol is 3 – 4 hours; 66% of the antibiotic being eliminated through urine and 15% through faeces.
- ◆ The autopsy of the test animals after 7 days from the experiment demonstrated that the intraperitoneal administration of ciprofloxacin is not irritating and does not cause adherences.

References

1. Chu DTW, Fernandes P. B., Structure – Activity Relationships of the Fluoroquinolones. Antimicrob Agents Chemother 1989; 33; 131 – 135.
2. Domagala J.M., Structure – Activity and structure - side – effect relationships for the quinolone antibacterials. J Antimicrob Chemother 1994; 33:685 – 706.
3. Rosentsiel N., Adam D.Quinolone Antimicrobials – An Update of Their Pharmacology and Therapeutic Use. Drugs 1994; 47: 872-901.
4. Fort F. L. Mutagenicity of Quinolone Antibacterials. Drug Safety 1992; 7: 214-222.
5. Echols R. M. The Selection of Appropriate Dosages for Intravenous Ciprofloxacin. J Antimicrob Chemother 1993; 31: 783-787.
6. Cyprofloxacin Hydrocloride in Physicians' Gen Rx 1993: II-480.
7. Ciprinol – viitor promiþãtor, speranþe îndeplinite. Leaflet KRKA Slovenia. 1999; 1-22.
8. Dryden m. S., Wing A. J., Phillips I. low Dose Intraperitoneal Ciprofloxacin for the Treatment of Peritonitis in Patients Receiving Continuous Amabulatory Peritoneal Dialysis (CAPD). J Antimicrob Agents 1991; 28: 131-139.
9. Campoli-Richards D. M., Monk J. P., Price A., Benfield P., Todd P. A., Ward A. Ciprofloxacin – A Review of its Antibacterial Activity, Pharmacokinetic Properties and Therapeutic Use. Drugs 1988; 35: 373-447.

ANTERO-POSTERIOR MEASUREMENT OF LUMBER SPINAL CANAL ON MIDSAGITTAL MRI IN KASHMIRI ADULTS

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Abstract: *Normal values for AP dimensions of lumbar spinal canal have been determined on midsagittal MRI scans in 100 Kashmiri adults. A careful study of spinal canal combined with prudent use of normal values should allow diagnosis of lumber canal stenosis to be made with a high level of confidence.*

Key words: *Stenosis, Spinal canal.*

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Introduction

Low backache is a common clinical problem. The etiology in many of these patients is narrowing of the lumbar canal. The incidence and implications of lumbar canal stenosis are ganing increasing attention. The values of the normal interpedicular and midsagittal diameters are different at the various levels of the lumbar spinal canal in individuals of the same race and differ at identical levels in individuals of various races. Alterations in the linear dimensions may be deduced from careful inspection of plain radiographs. A number of authors have discussed methods of measuring the spinal canal[1,2]. In the present study, an attempt has been made to establish the range of antero-posterior dimensions of lumber canal in Kashmiri adult population on midsagittal MRI scans.

Materials and Methods

Hundred young adults (44 males & 56 females) referred to Department of Radiodiagnosis for MRI scan of lumbar spine were included in the study. Patients with history of intermittent claudication were excluded from the study. The age of the patients ranged from 26-40 years. MRI of lumber spine was performed on 1.5 Tesla scanner with the patient in supine position. The midsagittal image was magnified and antero-posterior dimensions of spinal canal was measured at each level with the help of electronic calipers. The AP diameter was taken as the shortest midline perpendicular distance from posterior margin of vertebral body to inner margin of neural arch. In order to reduce error due to human element, measurement at each level was made twice and average was obtained. From these measurements, Mean deviation, Standard deviation and Tolerance limits were calculated for both the sexes combined. Results are summarized in Table 1. The tolerance limits are those within which the central 90% of normall population might be expected to fall. From the table, it can be seen that the sagittal diameter at L 1 is largest and sagittal diameter at L 5 slightly larger than at L 4 level.

Table 1: Mean deviations (MD), Standard deviations (SD) and 90% Tolerance limits for sagittal diameter of lumbar vertebrae on MRI

Vertebra	*MD*	*SD*	*90% Tolerance*	
			Min	*Max*
L 1	16.5	1.7	13.1	19.9
L 2	16.2	1.5	13.2	19.2
L 3	16.2	1.4	13.4	19.0
L 4	16.0	1.4	13.2	18.8
L 5	16.1	1.5	13.1	19.1

Discussion:

The importance of the radiographic measurement of the spinal canal was first emphasized in 1934 by Elsberg & Dyke[5] who established the normal range of interpediculate distance in the thoracic and lumbar spine and applied this to the diagnosis of intra-spinal tumors. These early works concerning the large spinal canal stimulated interest in the small canal. Though Sarpyener[6] is credited with first recoognizing the clinical importance of lumbar canal stenosis, Verbiest[7] was first to assess stenosis quantitatively. He classified stenosis as relative when the sagittal diameter is 12 mm and absolute when the diameter is 10mm or less[8].

However, the value of the lowest normal Interpedicular and midsagittal diameters is different in the various levels of the lumbar vertebral canal of individuals of the same race and differ at identical levels in individuals of various

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races. Poostacchini petal in a morphometric study of 121 skeletons (63 Italians and 58 Indians) found that the mean AP diameter to be significantly greater in Italian skeletoons. Hinck et al[4] measured the AP diameter of the spinal canal on plain radiographs in western population. The mean AP diameter in his study of 49 adults was 21.8 mm at L 1, 21.7 mm at L 2, 21.5 mm at L 3, 21.6 mm at L 4, and 21.4 mm corresponding levels in our study.

References:

1. Janjua M Z, Muhammad F. Measurement of normal adult lumbar spinal canal. J Pak Med Assoc 1989;399(10):264-8.
2. Gupta S K, Roy R C, Srivastava A. Sagittal diameter of cevvi-cal canal in normal Indian adults. Clin Radiol 1982;33(6):681-5.
3. Benjamin C P Lee, Elias Kazam, Arnold. D. Computed Topography of the spine and spinal cord. Radioloogy 1978;128:95-102.
4. Vincent C Hinck, Carl E Hoopkins, William M. Clark. Sagittal diameter of the lumbar spinal canal in children and adults. Radiology 1965;85:929-937.
5. Elsberg CA, Dyke C G. Diagnosis and localization of tumors of spinal coord by means of measurements made on X-ray films. Bul Neurol Ist NY 1934;3:359-393.
6. Sarpyener M A. Congenital stricture of the spinal canal. J Bone and joint surgery 1945;27:70-79.
7. Verbiest H. Further experiences on the pathologcal influence of developmental narrowness of bony lumbar vertebral canal. J bone and joint surgery 1955;37B:576-583.
8. Verbiest H. Neurogenic intermittent claudication with special reference to stenosis of the lumbar vertebral canal. Amsterdam: North Holland 1976.

MASSIVE PULMONARY EMBOLISM DURING PREGNANCY WITH ANTIPHOSPHOLIPID ANTIBODY SYNDROME SUCCESSFULLY MANAGED WITH SYSTEMIC THROMBOLYSIS

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ABSTRACt: *Venous thromboembolic disease (VTE), including deep vein thrombosis and pulmonary embolism is not uncommon during pregnancy. Many clinical features of VTE viz. leg swelling, increased respiratory rate etc., being common during normal pregnancy, a high degree of suspicion needs to be maintained for diagnosis of pulmonary embolism(PE). Furthermore, anticoagulants and systemic thrombolytic agents used for the treatment of pulmonary embolism have their own limitations.*

We report a case of massive pulmonary embolism, during twenty eighth week of pregnancy, sucessfully treated with systemic thrombolysis.

Key Words: *Pregnancy. Pulmonary Embolism, Thrombolysis*

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INTRODUCTION

Normal pregnancy being a physiological hypercoagulable state predisposes pregnant females to deep venous thrombosis (DVT). In addition, pregnancy can unmask an underlying hyprecoagulable state viz., Factor V Leiden mutation or antiphospholipid antibody syndrome (APLAS).

DVT carries a significant risk of pulmonary embolism which can have catastrophic consequences for mother and foetus. Diagnostic tests (venous doppler, echo, spiral CT, ventilation perfusion scan (V/Q scan) and pulmonary arteriography) need to be carefully chosen and dictated by the clinical situation, availability of tests and expertise.

Case report

A 28 yrs old female para 2, gravida 1 presented in 8th month of pregnancy with history of right sided chest pain, shortness of breath and postural syncope. The symptoms were of two days duration. Past history revealed one abortion. On examination she was tachypnoeic with oxygen saturation of 86% on room air: Pulse rate was 140 beats/mt. and right upper limb blood pressure was 80/40 mmHg. Her jugular venous pressure was elevated with prominent v waves. Cardiovascular examination revealed clinical signs of pulmonary arterial hypertension with tricuspid regurgitation and right ventricular S3 gallop. Chest examination revealed signs of a small pleural effusion on right side. Pulmonary embolism was suspected as there was no obvious reason for her clinical picture. Twelve lead electrocardiogram(ECG) (Figure 1), taken at admission, revealed sinus tachycardia, S 1 Q3T3 patten, ST elevation in leads Vl and AVR. Plasma D-dimer levels were 1000ngms/ml (normal levels upto 500 ngms/ml). Echocardiography revealed dilated right atrium, right ventricle and main pu-lmonary artery. Interatrial and interventricular septae were intact and pushed to left. There was paradoxical motion of the interventricular septum. Doppler interrogation revealed moderate tricuspid regurgitation with expected pulmonary arterial systolic pressure of 50mmHg (Fig. 2). Pulmonary perfusion scan (Fig.3) revealed perfusion defects involving most of right lung and multiple segments of left lung, findings consistent with massive pulmonary embolism (PE).

She was treated with intravenous tissue plasminogen activator (tpA) 100 mgs given over 90 minutes. She also received intravenous heparin (bolus and infusion). Immediately after thrombolysis patient had a dramatic clinical improvement. Her blood pressure improved to 110/80 mmHg, right ventricular gallop disappeared and oxygen saturation on room air improved to 96%. No untoward effects were noted and she made a good clinical recovery. In her subsequent ECGs and echocardiographic studies the signs of right ventricular strain showed complete resolution.

Her ESR, CRP, protein C, protein S, antithrombin III and activated protein C levels (done before starting heparin) were normal. Other investigations including complement, ANA, C-ANCA and p-ANCA were also normal. Her

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antiphospholipid antibody levels (IgG) were moderately elevated. Peripheral vascular Doppler study did not reveal any DVT.

Discussion

Normal pregnancy is a physiological hypercoagulable state characterized by increased levels of vitamin K – dependent clotting factors, factor VIII and Vonwillebrand's factor. Additionally fibrinolytic activity is depressed. This hypercoagulable state coupled with venous stasis resulting from gravid uterus increases the risk of venous thrombosis.

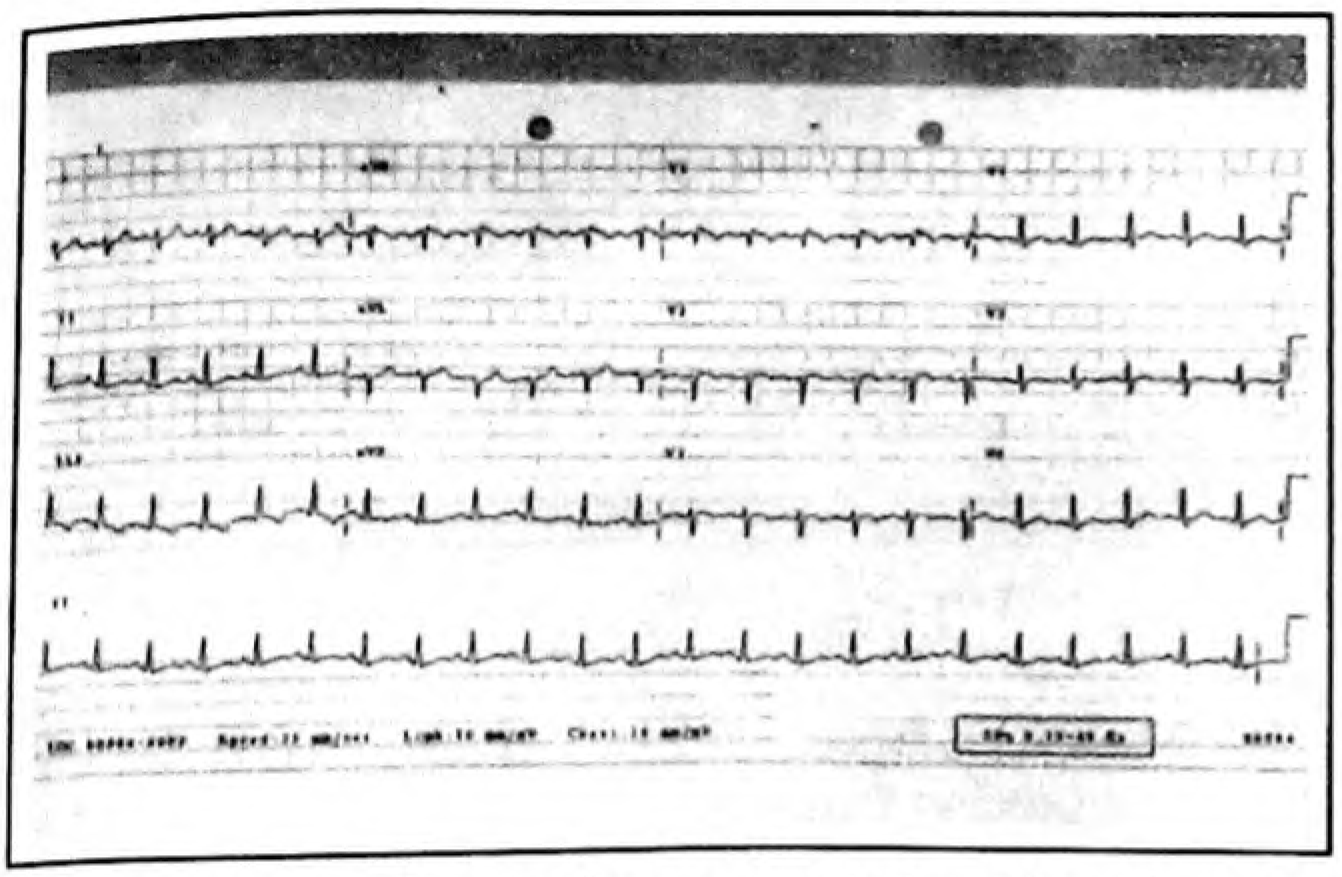

Fig. 1: Electrocardiagram showing sinus tachycardia, right axis deviation S1 Q3 T3 pattern and ST segment elevation in leads V1 AVR

DVT is reported to complicate 0.21% of pregnancies and nearly half of them present in the second trimester of pregnancy. In 82% one or both lower limbs are involved. Pulmonary embolism occurs once in seven hundred and fifty pregnancies. Antenatal deep venous thousand embolism (DVT) tends to be left sided and involves the ileofemoral system (1,2,3).

Pregnancy can unmask antiphospholipid antiody syndrome(APLAS). The latter is characterized by antibodies directed against various phospholipid containing proteins like glycoprotein IIb, Annexin V, protein C and protein S. In addition they may induce expression of various adhesion molecules like E-selection, VCAM-I and ICAM-I. This promotes monocyte adhesion to endothelial cells precipitating vascular thrombosis(4-6). APLAS may account for 15-20% of all episodes of DVT, one third of all episodes of stroke in patients less than 50 yrs of age and 5-15% of females with recurrent foetal loss(4). Antiphospholipid antibodies can be detected with clotting assays (lupus anticoagulant) or immunoassays (anticardiolipin antibodies).

Electrocardiogram is done to rule out myocardial infarction and to identify a large pulmonary embolism.

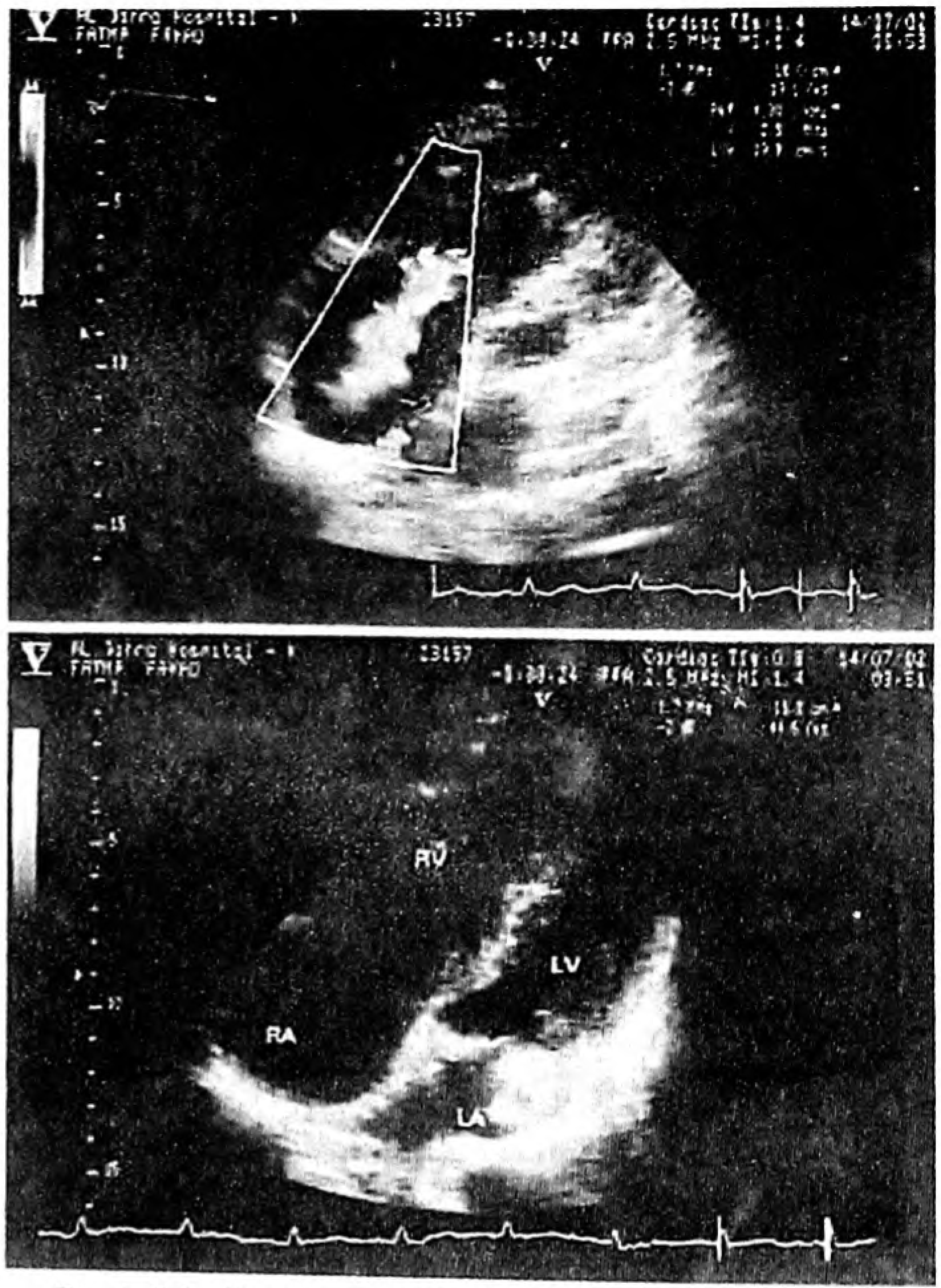

Fig. 2: Echo Doppler study revealing dilated right atrium (RA), right ventricle (RV) and inter atrial septum pushed to left. Colour flow mapping (CFM) revealing moderate tricuspid regurgitation

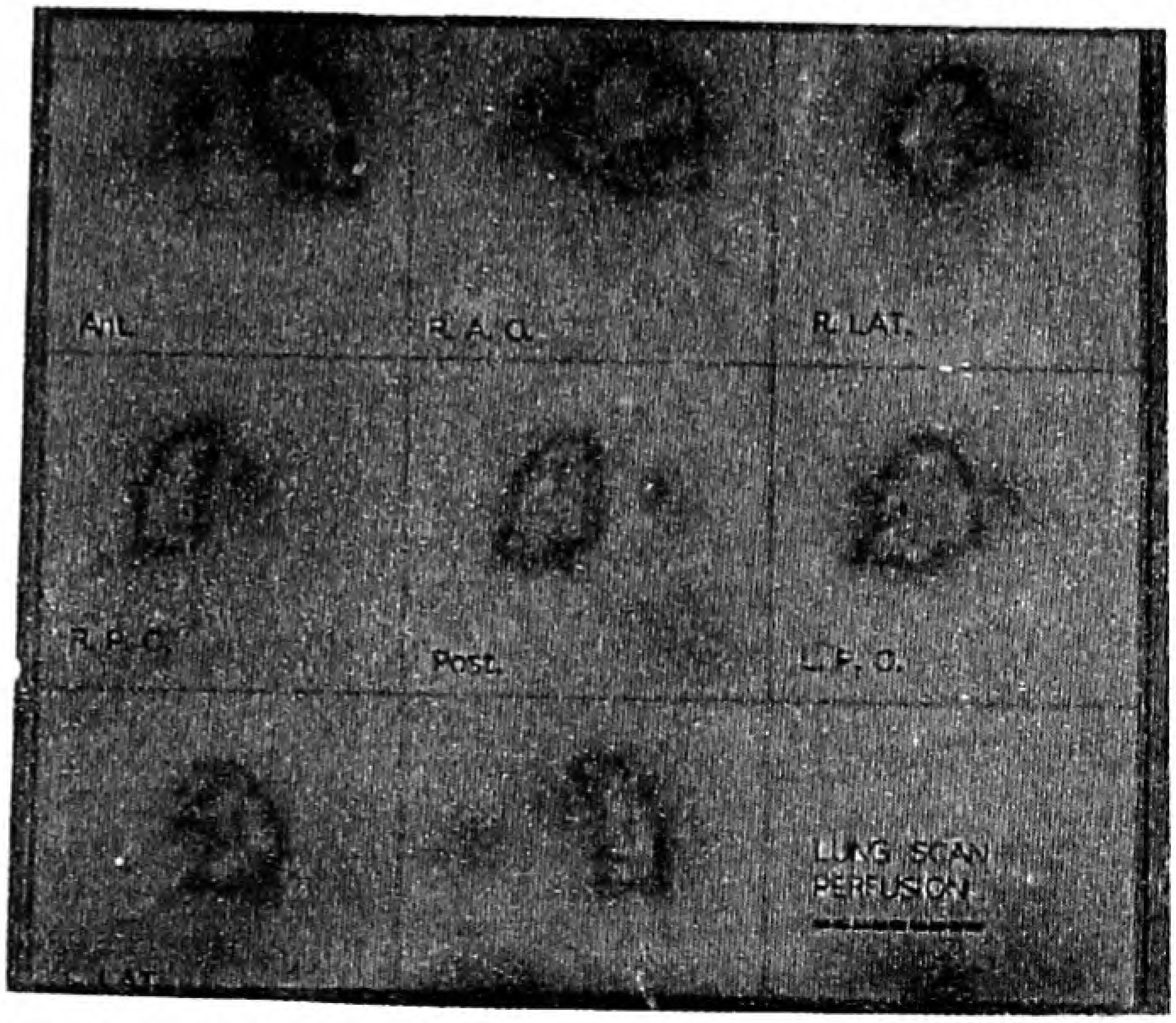

Fig. 3: Perfusion Lung Scan revealing lung perfusion defect in right lung and multiple small defects in left lung.

Plasma D-dimer levels are important for screening. In some centres after clinial suspicion of PTE, plasma D-dimer levels and CXR are routinely obtained. In case both these tests are negative, the diagnosis of PTE is considered excluded at that point of time(17).

While as echo is insensitive for diagnosis of PTE, it is an important tool for triage. It has been recently shown that the subgroup of PTE patients with a positive echo study viz (RV dilatation, IVC plethora, RV free wall hypokinesia with apical sparing, tricuspid regurgitation with pulmonary arterial hypertension) had a mortality of around 8%, despite normal systemic blood pressure(20).

Exposure to radiation even after completion of organogenesis may cause growth retardation, CNS abnormalities or even childhood cancers. However less than 5 rads exposure during pregnancy is reassuringly safe(9). Pulmonary scintigraphy is associated with a fetal exposure of less than 0.05 rads.

DVT or small PTE may be treated with systemic anticoagulation with unfractionated or low mol. weight heparin(10). Massive or submassive PE(11) is a medical emergency and therapeutic options should be cautiously individualized. In absence of contraindications systemic or catheter directed thrombolysis should be attempted. In case of contraindication, surgical thrombectomy is an option.

Pulmonary embolism poses a therapeutic challenge in pregnant patients because of possible risk of bleeding and fetal damage due to thrombolytic therapy(12-20). Furthermore, Monitoring heparin efficacy in pregnancy can be problematic especially high doses of heparin are required. It has been recommended that in patients with such a situation plasma anti Xa levels be monitored. Additionally anti Xa level assay have been found to be useful for judging efficacy of LMWH and in patients with APLAS who require unfractionated heparin or LMWH(8). In cases with recurrent PTE, IVC filters are recommended. Anticoagulants are continued despite the device in place(21).

REFERENCES

1. Metha AB, Hoffbrand AV. Haematological aspects of systemic disease. In Ho6brand AV. Ed. Postgraduate Hematology, Fourth Edition. Butterworth – Heinemann-Oxford. P687-688,1997.
2. Ray JG, Chan WS. Deep vein thrombosis during pregnancy and the puerperium; ameta-analysis ofthe period of risk and the lack of presentation. Obstet Gynecol Surv. 1999 April, 54 (4): 265-71.
3. Lee RV, McComb LE, Messadri FC. Pregnant patients, painful legs: The Obstetricians dilemma. Obstet Gynecol Surv 1990;45: 290-298.
4. Asherson RA, Khamshta MA, Osdio-Ros J et al. The primary antiphospholipid syndrome, Medicine 1989;68:366-74.
5. Petri M. Pathogenesis and treatment of the Antiphospholipid antibody syndrome. Med. Clinic North Am. 1997;81:151-77
6. Lechnes K, Pabinges - Fasching I. Lupus anticoagulants and thrombosis: A study of 25 cases and review of literature. Haemostasis 1985;15:254-62.
7. McConnell MV, Solomon SD, Rayon ME et al. Regional right ventricular dysfunction detected by Echocardiography in acute pulmonary embolism. Am J Cardio 1993;18:585.
8. Goldhaber SZ. Pulmonary embolism pp 1896-1907 Braunwald E et al (eds): Heart disease (6th edition) USA Saunder 2001.
9. Elkayam U. Pregnancy and cardiovascular disease. In: Braunwald E et al (eds): Heart Disease 6th edition USA Saunders 2001, pp 2172-90.
10. Chan WS, Ray JG. Low molecular weight heparin use during pregnancy: issues of safety and practicality. Obstet Gynecol Surv 1999 Oct.:54(10): 649-54.
11. Konstantindes S, Gerbel A. Heusel G et al. Heparin plus alteplase compared with heparin alone in patients with submassive pulmonary embolism. N Engl J Med 345(15):1143-1150.
12. Rinaldi JP, Yassine M, Aboujaoude F, et al. Successful thrombolysis on aortic valve prosthesis by plasminogen tissue activator during pregnancy (French). Arch Mal Cocur Vaiss 1999 April: 1992(4): 427-30.
13. Janssens U, Klues HG, Hanrath P. Successful thrombolysis of right atrial and ventricle thrombi in a patient with peripartum cardiomyopathy and extensive thromboembolism. Heart 1997 Nov.: 78 (5): 515-6.
14. Nizwa J, OhyamaH, Matumura S, Maeda Y, Shimisu T. Treatment of acute superior sagittal sinus thrombosis by t-PA infusion via venography- direct thrmbolytic therapy in the acute phase. Surg Neurol1998 April:49(4): 425-9.
15. Weber MD, Halligan RE, Schumacher JA. Acute infraction intracoronary thrombolysis and primary PTCA in pregnancy. Cathet Cardiovasc Diagn 1996;42:38-43.
16. Schumacher B, Belfort MA, Card RJ. Successful treatment of acute myocardial infarction during pregnancy with tissue plasminogen activator. Am J. Obstet Gynecol 176:716 -719-1997.
17. Goldhaber SZ. Contemporary pulmonary embolism thrombolysis. Chest 107:45S;1991
18. Meneveau N, Schiele F, Metz D, et al. Comparative efficacy of a two hour regimen of streptokinase versus alteplase in acute massive pulmonary embolism: Immediate olinical and hemodynamic outoome and one year follow up. J Am Coll Cardiol 31:1057;1998.
19. Tebbe U. Graf A, Kamke W et at. Hemodynamic effects of double bolus reteplase versus alteplase infusion in massive pulmonary embolism. Am heart J 138:39;1999.
20. Ginsberg JS, Jack H. Use of antithrombotic agents during pregnancy. Chest 114/ 5/November 1998; supplement 5245-5305.
21. Bali HK, Banerjee S, Aggarwal M, Sharma SK, Bahl A, Mohi-ud -din Khalid, Vergiya RV. Percutanous IVC filter in recurrent pulmonary thromboemsolism. Bull PGI, 1999;33:37-40.

CONGENITAL BILATERAL BRACHYMETATARSIA-LENGTHENING BY DISTRACTION OSTEOGENESIS

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ABSTRACT: Brachymetatarsia is not an uncommon condition. Lengthening by distraction osteogenesis is described in a 26-year old girl with congenital bilateral Brachymetatarsia. Lengthening was performed using UMEX distractor and the desired length was achieved in both metatarsals at an average healing index of 1.2 months /cm. Pin tract infection was the only problem encountered during the process of lengthening. Distraction osteogenesis is an effective method of correction of brachymetatarsia.

***Key words:** Brachymetatarsia, Ilizarov technique, distraction osteogenesis*
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Introduction and Review of Literature

Brachymetatarsia is not an uncommon deformity resulting from premature closure of epiphyseal plate. 4th metatarsal is commonly involved and the bilateral occurrence has been reported to be 72%[1]. An incidence of 1 in 1,820 and 1 in 4,586 with a male to female ratio of 4: 98[1] has been reported in the literature[1]. Brachymetatarsia may be associated with a number of systemic conditions[2]. Clinically short metatarsal results in corresponding shortening of the digit and a floating toe. There is excessive load on other metatarsals, which may result in the formation of painful plantar hyperkeratotic lesions. Patients are concerned primarily about the appearance of their feet and may also present with difficulty in walking and problems with shoe ware. Most of the patients seek treatment for cosmetic reasons.

In 1969 McGlamry and Cooper[3] were the first to report surgical correction with autogenous bone graft from calcaneus. Since then various techniques of single stage osteotomy and bone grafting have been reported with satisfactory results[4, 5]. Later modifications differ in the source of bone graft like the use of navicular[6] or use of proximal phalyngeal heads[7]. Urano and Kobayashi[1] used interposition of spindle shaped graft in between metatarsophalyngeal joint, however compromising the joint function. Procedures combining lengthening of short metatarsal by allogenic bone graft and shortening of other metatarsals has also been described[8] Mah and colleagues reported use of Calnan Nicolle implant in the metatarsophalyngeal joint as a spacer[9]. The disadvantages of implant rejection and aseptic necrosis limit its use in young patients Techniques involving slide lengthening of metatarsals have also been reported in the literature[10]. After the introduction of principles of distraction osteogenesis by G A Ilizarov, lengthening by gradual distraction without bone grafting gained much importance[11]. The technique provides the advantages of simplicity of technique and simultaneous and gradual lengthening of soft tissues without damage to neuro vascular tissues. We present a case of bilateral metatarsal lengthening by distraction osteogenesis with an excellent result.

Case report

A 26 years old girl presented to us with bilateral congenital short 4th metatarsals. Patient felt concerned because of the ugly appearance of her feet. Examination of the feet revealed short 4th toe on either side, overriding the adjacent toes. There were no callosities. Radiography revealed bilaterally symmetrical shortness of 4th metatarsals on either side measuring 3 cms. One foot was operated at a time starting with the left foot. Surgery was under taken under regional block and tourniquet control using UMEX distracter. The metatarsal was approached through a dorsal one cm incision. The extensor tendon was retracted to one side and a low energy osteotomy performed towards the base of the metatarsal. This was followed by fixation of one threaded pin, each in the proximal and the distal fragment and their incorporation in the distracter. Patient was discharged next day after teaching distraction and pin care. Distraction was started on the 7th day at the rate of 1/4th of nut turn three times a day. Distraction rate was adjusted according to the appearance of regenerate on X – rays and was continued till the desired length was achieved. During the lengthening and consolidation period

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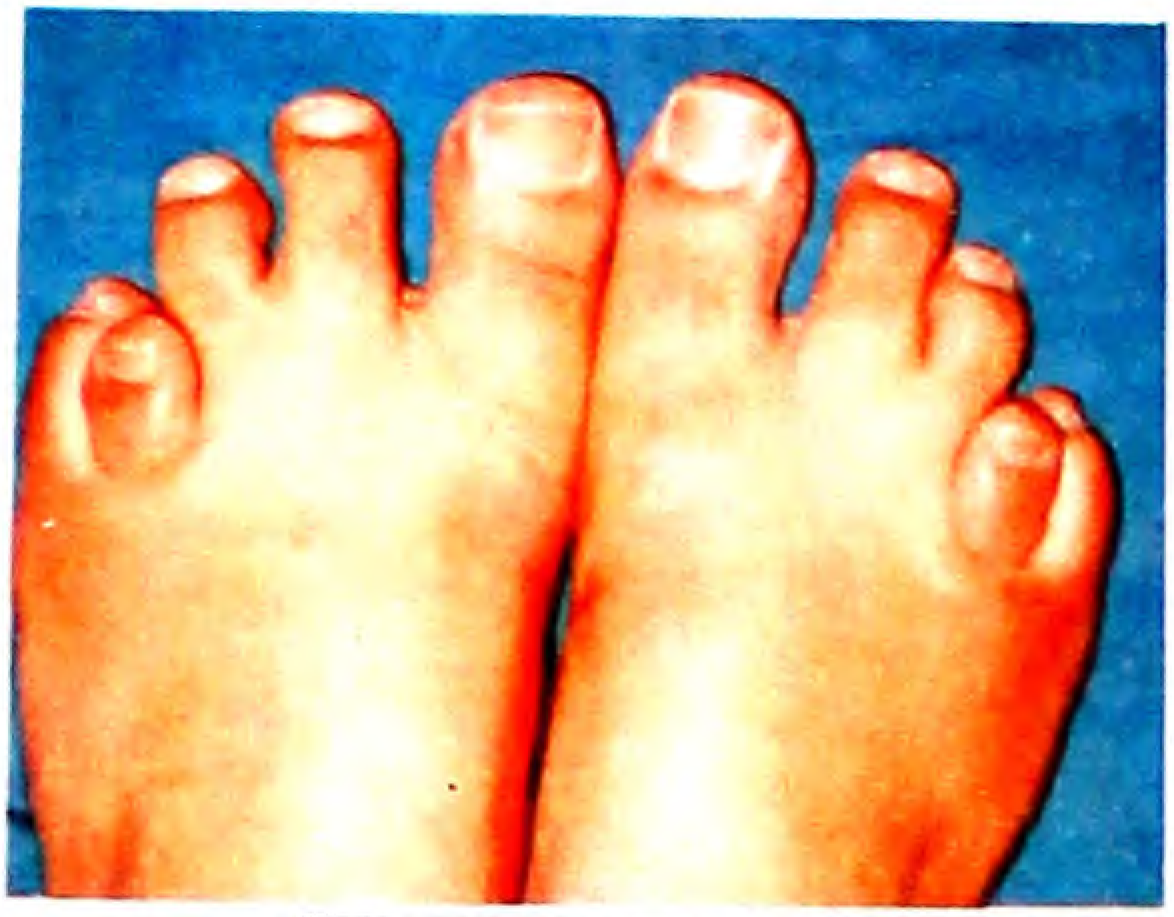

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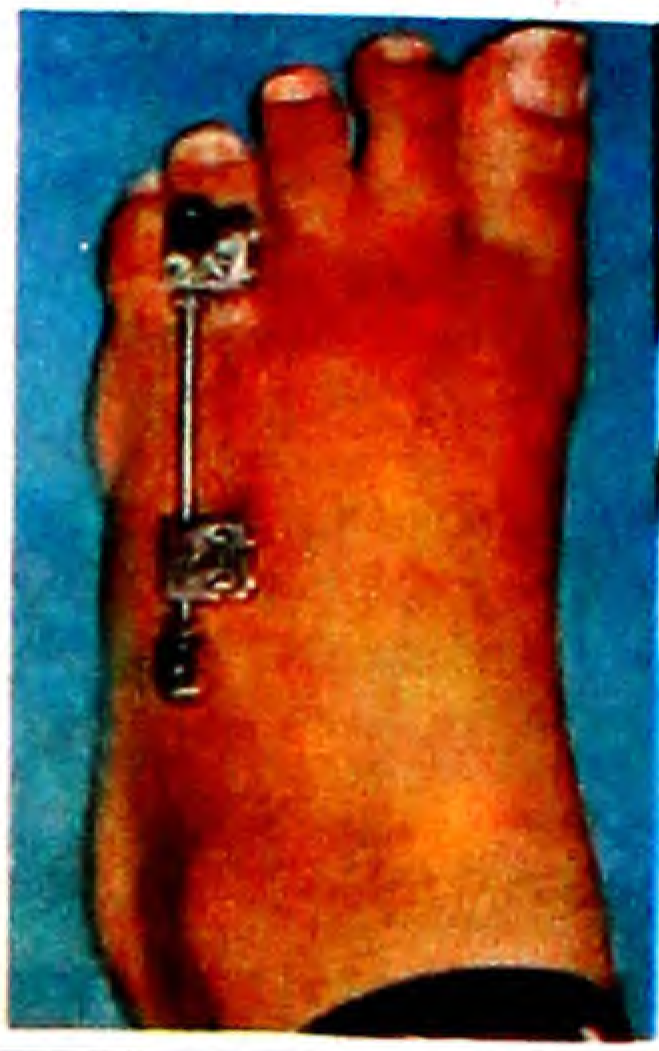

Fig 5↑

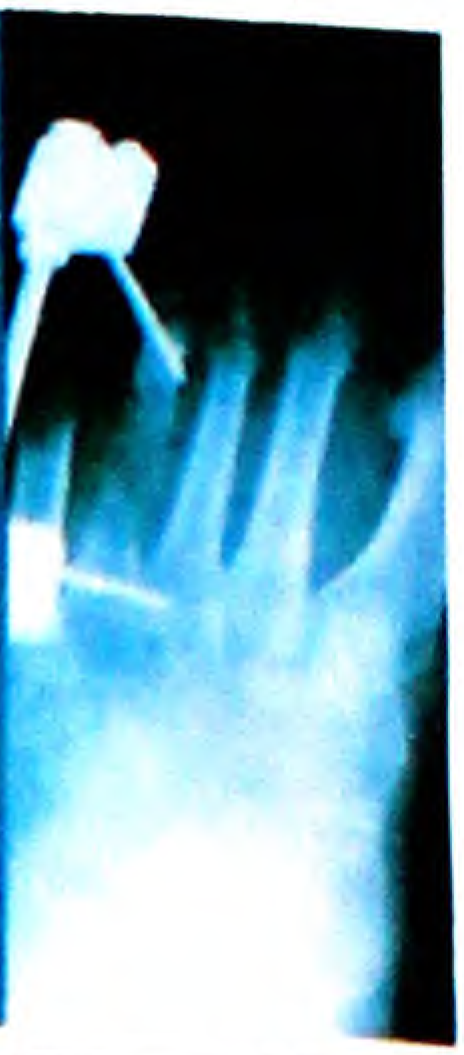

Fig 6↑

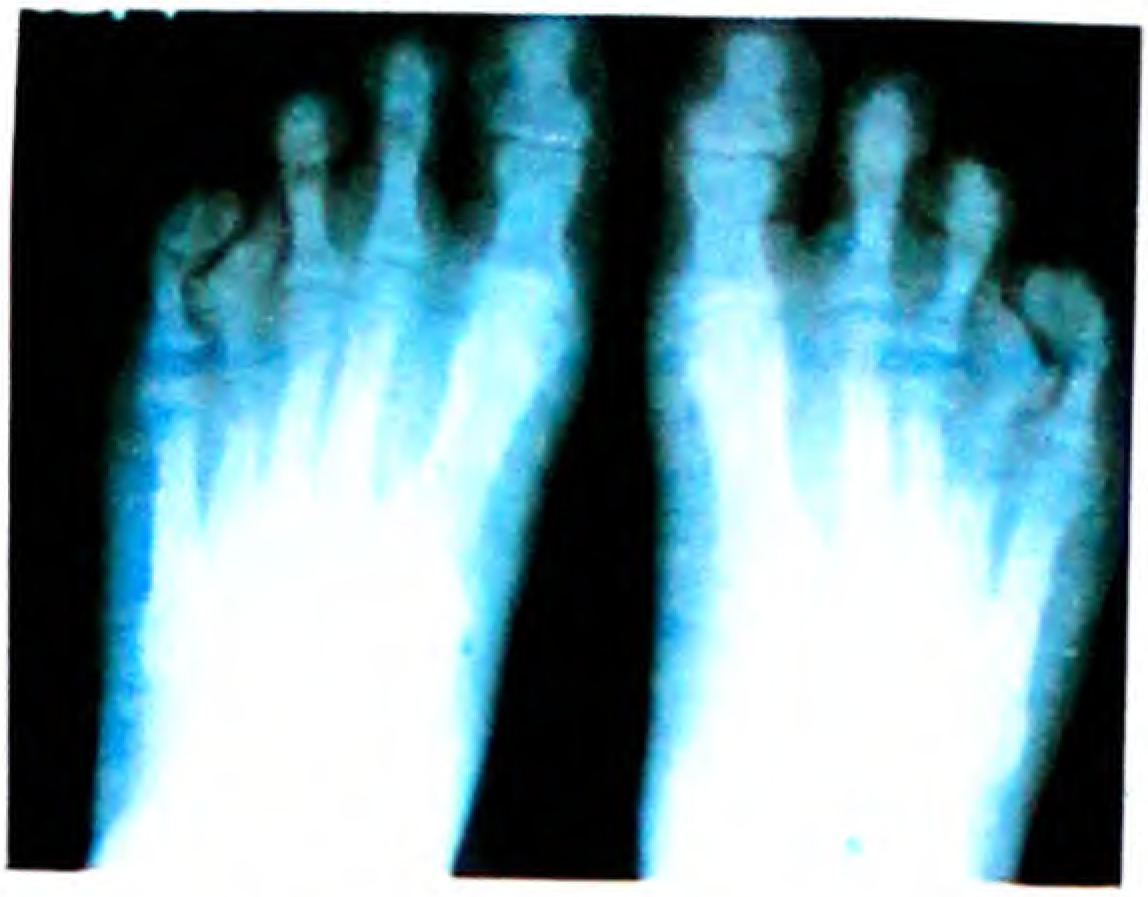

Fig. 2 ↑

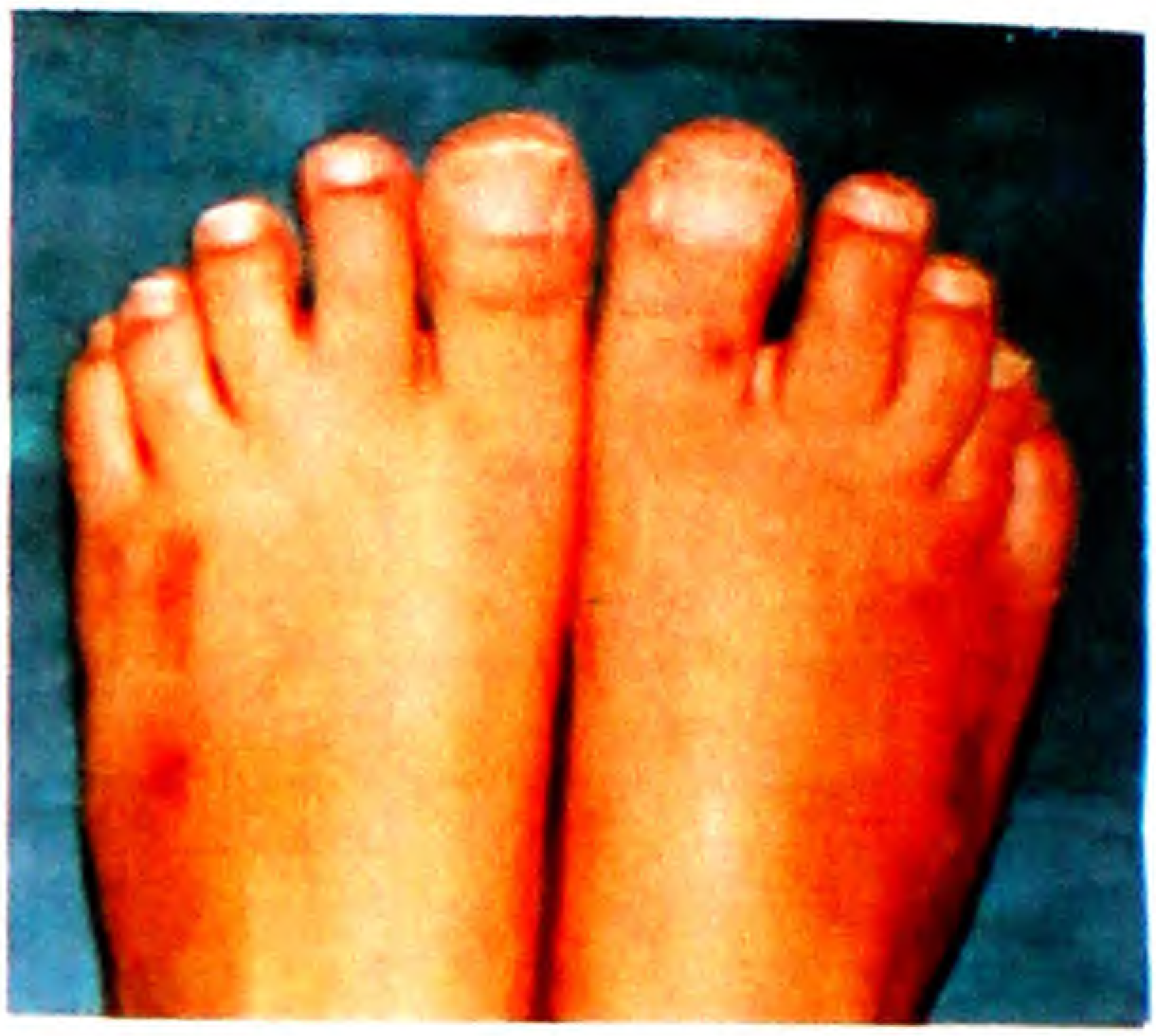

Fig. 7↑

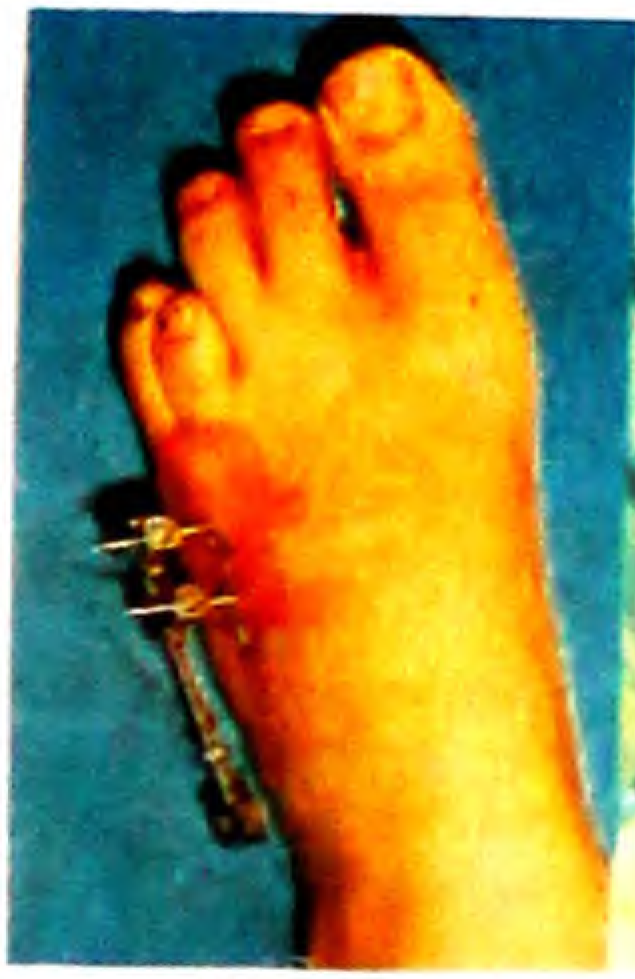

Fig. 3 ↑

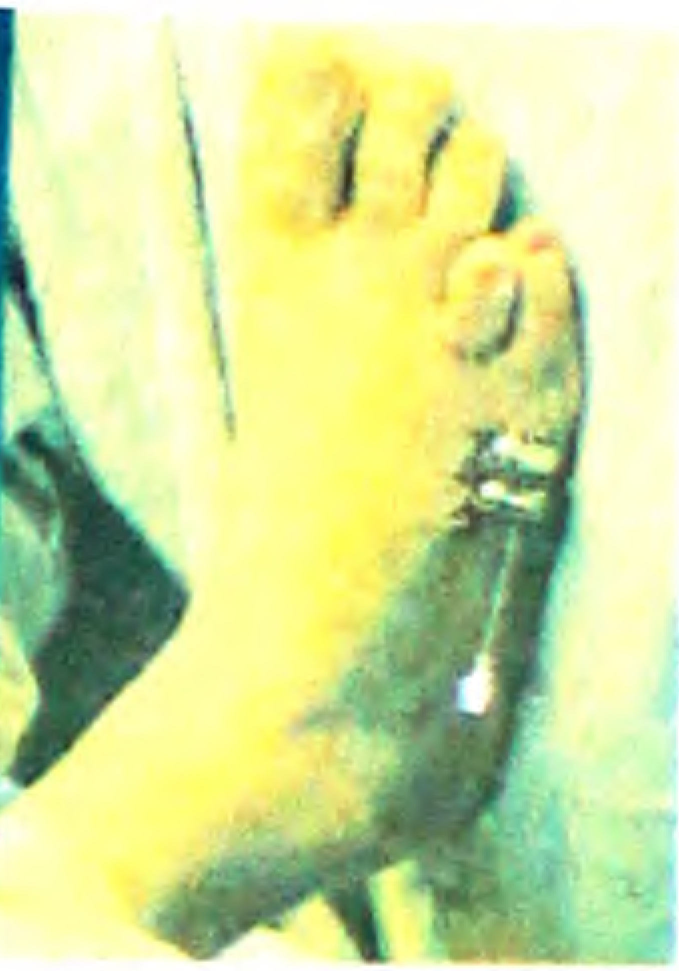

Fig 4↑

Legends

Fig.1	Pre operative photograph showing bilateral 4th toe shortening
Fig.2	X Ray showing bilateral 4th metatarsal shortening.
Fig.3	Postoperative photograph showing distracter applied on left foot.
Fig. 4	Postoperative photograph showing distracter applied on right foot.
Fig. 5	Photograph showing length equalized.
Fig. 6	X ray showing new bone formation.
Fig. 7	Photograph showing final result.

the patient was allowed to walk on the heel on the operated side. The distracter was removed when corticomedullary differentiation was found on the X rays. The other foot was similarly treated after a gap of 3 months. Both toes were symmetrically lengthened and the average length gained on either side was 3cms.Both metatarsals healed with an average healing index of 1.2 months/cm. There were no major complications during the whole process of lengthening. Mild pain during the lengthening phase was the only problem encountered. Patient was satisfied with the final result and the improved appearance of her feet.

Discussion

The use of distraction osteogenesis for metatarsal lengthening was safe and very effective in our patient. Except for a mild pin tract infection, there was no major complication. In contrast to the Orthofix mini fixator, we used only two small diameter pins in the UMEX mini fixator to prevent scar formation and thus achieve better cosmesis after surgery. We did not come across any loss of fixation or any angulation deformity during lengthening. The assembly became more stable after distraction was started.

Techniques using lengthening by acute distraction and the use of bone graft have a number of disadvantages notably potential neuro vascular compromise, non-union, pseudoarthosis, the limited amount of length that can be achieved and the donor site morbidity[12]. Present day methods of metatarsal lengthening which have been shown to provide good results include the techniques of single stage lengthening using gradual intra operative distraction with bone grafting and gradual distraction by callotasis. Goo Hyun Back et al[13], using gradual distraction intra operatively over 20 to 30 minutes to prevent neuro vascular damage and the use of K wires to prevent metatarsophalyngeal joint subluxation presented good results in their series of 34 metatarsal lengthening. The average length of 14 mm gained, however is lesser than reported in the series using callotasis[14]. In Ho Choi et al, comparing the results of 15 one stage lengthening and bone grafting with 9 metatarsal lengthening by callotasis found no major differences in terms of length gain, percentage increase in length and complications but found earlier consolidation in the one stage lengthening group than in the callotasis group[14]. They however concluded that callotasis should be preferred in patients requiring larger and multiple lengthening. There are many reports of metatarso phalyngeal subluxation with larger lengthening using callotasis. Masada in his series concluded that lengthening by callotasis should not exceed 40% of the original length of the metatarsal[15]. Subluxation was found in two patients in Takao's series that required lengthening greater than 40 % of the original length of the metatarsal[16]. We achieved an average lengthening of 30 mm without any subluxation, non-union or vascular compromise. We feel that achievement of larger length and lack of donor site morbidity by callotasis could over weigh the disadvantages of delayed consolidation. Further more, distraction can be stopped or even reversed if metatarsophalyngeal joint dislocation is suspected. However more reports of larger lengthening achieved by callotasis without metatarsophalyngeal dislocations are required to substantiate this view.

References

1. Urano Y, Kobayashi A: Bone lengthening for shortness of the fourth toe. J Bone Joint Surg 1978; 60 A: 91 – 93.
2. Oreenfield OB: Radiology of Bone diseases, 3rd ed. Philadelphia, J. B. Lippincot, 1980, p. 344.
3. McGlamry ED, Cooper CT: Brachymetatarsia: A surgical treatment. J Am Podiatr Assoc 1969; 59: 259 – 264.
4. Kaskuk KB, Hanft JR, Schabler JA, Kopelman J: Alternative autogenous bone graft donor sites in Brachymetatarsia reconstruction: A review of the literature with clinical presentations. J Foot Surg 1991; 30: 346 – 252.
5. Jimenez Al: Brachymetatarsia: a study in surgical planning. J Am Podiatr Assoc 1979: 69: 245 – 251.
6. Pasternack WA: Brachymetatarsia. A unique surgical approach. J Am Podiatr Med Assoc 1988; 78: 415 – 418.
7. Rock GD, Gaspari C, Mancuso JE: Brachymetatarsia with the use of digital arthoplastic bone. J Foot Surg1993; 32: 499 – 504.
8. Handelman RB, Perlman MD, Coleman WB: Brachymetatarsia: A review of the literature and case report. J. Am Podiatr Med Assoc1986; 76: 413 – 416.
9. Mah KS, Beagle TR, Falkner DW: A correction for short fourth metatarsal. J Am Podiatr Assoc 1983; 73: 196 – 200.
10. Tabak B, Lefkowitz H, Steiner I: Metatarsal slide lengthening without bone grafting. J Foot Surg 1986; 25: 50 - 53
11. Ilizarov G. A. The tension Stress effect on the genesis and growth of tissues Part 1; the influence of stability and fixation and soft tissue preservation. Clinic. Orthop, 1989, 238: 249 – 281,
12. Bouchard JI. Congenital deformities of the forefoot. In: McGlamery ED, Banks AS, Darmy MS, Eds, Comprehensive textbook of foot surgery. Vol. 2. Baltimore, etc: Williams and Wilkins 1992: 1191 – 231.
13. Baek GH, Chung MS. The treatment of congenital brachymetatrsia by one stage lengthening. J Bone Joint Surg (Br) 1998; 80 B 1040 – 4.
14. Choi IH, Chung MS, Baek GH, Cho TJ, Chung CY. Metatarsal lengthening in congenital brachymetatarsia: one-stage lengthening versus lengthening by callotasis. J Pediatr Orthop 1999 Sep-Oct; 19(5): 660-4
15. Masada K, Fujita S, Fuji T, Ohno H. Complications following metatarsal lengthening by callus distraction for brachymetatarsia. J Pediatr Orthop 1999 May-Jun; 19(3): 394-7
16. Kawashima T, Yamada A, Ueda k, Harii K: Treatment of Brachymetatarsia by callus distraction (Callotasis). Ann plast Surg 1994; 32: 191 – 199.

ACUTE POSTERIOR MULTIFOCAL PLACOID PIGMENT EPITHELIOPATHY

Tariq Qureshi M.S.; Ghulam Rasool MS; Hans Raj Sharma MS; Salima Dar MS

***ABSTRACT**: The clinical and fluorescein angiographic findings are described in a young female patient showing bilateral acute loss of vision secondary to multifocal,yellow- white,placoid lesions at the level of the pigment epithelium; Rapid resolution of these lesions and significant improvement in visual acuity without any treatment. The visual disturbance occurred after a flu-like illness.*

***Key Words** : APMPPE, Retinal Pigment epithelium.*

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INTRODUCTION

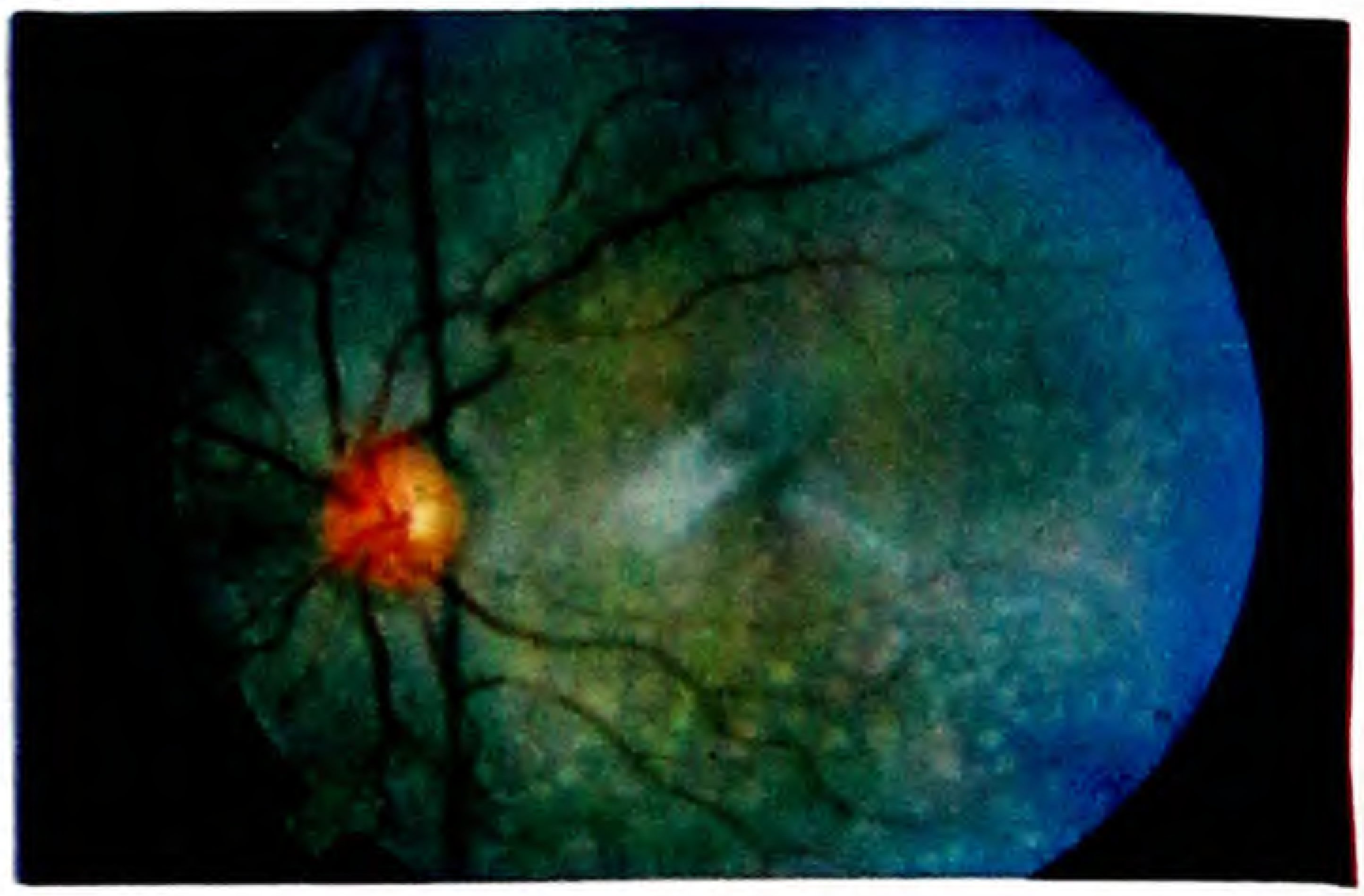
Acute posterior multifocal placoid pigment epitheliopathy (APMPPE) originally defined by Gass in 1968 , is characterized by the acute onset of bilateral multifocal, yellow-white placoid lesions of the posterior pole at the level of the retinal pigment epithelium. These resolve spontaneously leaving extensive derangement of the pigment epithelium but minimal alteration of the choroid or retina. When located in the macular area, the lesions cause a rapid reduction of visual acuity. Visual acuity spontaneously improves in most cases over several weeks and improvement may continue even months after visible inflammatory activity has ceased.

In the acute stage, the fluorescein angiographic appearance of the condition is characterized by placoid lesions blocking transmission of the choroidal fluorescence in the initial phase of the angiogram, which are gradually stained and fluoresce faintly in the late phase. Older lesions in various stages of resolution represent residual defects in the pigment epithelium and appear as transmission or window - type defects without late staining or leakage of dye. No etiological factor could be found despite extensive general physical examination and blood studies. Most often ,there is history of viral fever prior to the onset of visual disturbance.

CASE REPORT

A 21 year old female complained of a three - day history of decreased vision in both eyes. She also gave a history of flu – like illness consisting of low – grade fever, headache, generalized malaise, muscle - aches and mild cough two weeks prior to the onset of visual disturbance. These symptoms lasted about 7 days.

Examination revealed a best corrected visual acuity of 6/36 in both eyes. External examination, pupillary response, and motility were normal. Slit - lamp examination was also normal in both eyes.



Ophthalmoscopic examination showed multiple , small, yellow-white placoid subretinal lesions with fuzzy margins scattered throughout the posterior pole. The disks were hyperaemic with well defined margins.

Fluorescein angiography revealed early blockage of the choroidal pattern with late staining.

Laboratory investigations including chest roentgenogram, complete blood cell count (CBC), ESR, urine, blood culture, blood sugar test, tuberculin test and

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serological examination were all unrewarding.

A diagnosis of APMPPE was made, and no therapy was prescribed. When examined four weeks later, her vision was 6/9 bilaterally and most of the placoid lesions had disappeared. Mottling of the macular pigment was still evident.

COMMENT

This case illustrates several common findings of APMPPE , i.e. a young patient with a prodromal viral - type syndrome, typical bilateral fundus lesions, characteristic fluorescein angiography and noncontributory medical work-up. In addition, there was macular pigment mottling, which has also been reported in other series.

The detailed description, provided by Gass (1968) and others , of this specific reaction pattern enables it to be distinguished from other inflammatory and degenerative choroido – retinal affections. Whatever its cause, the disease appears to be clinically benign, requires no treatment, is self limited, and usually resolves within a matter of weeks, leaving normal vision but a grossly disturbed pigment epithelium. So, once the diagnosis is made, the patient can be informed, even in the acute stage of near blindness, of the relatively benign prognosis.

REFERENCES

1. Gass JDM : Acute posterior multifocal placoid pigment epitheliopathy. Arch Ophthalmol 80: 177 - 185, 1968.
2. Fitzpatrick PJ, Robertson DM :- Acute posterior multifocal placoid pigment epitheliopathy. Arch Ophthalmol 89: 373 - 376, 1973.
3. Deutman AF, Oosterhuis JA, Boen - tan TN, Aan de Kerk AL:- Acute posterior multifocal placoid pigment epitheliopathy. Br J Ophthalmol 56: 863 - 874,1972.
4. Annesley WH,Tomer TL, Shields JA:- Multifocal placoid pigment epitheliopathy. Am J Ophthalmol 76: 511 - 518, 1973.
5. Savino PJ, Weinberg RJ, Yassin JG, Pilkerton AR:- Diverse manifestations of acute posterior multifocal placoid pigment epitheliopathy. Am J Ophthalmol 77: 659 - 662, 1974.
6. Ryan SJ, Maumenee AE:- Acute posterior multifocal placoid pigment epitheliopathy. Am J Ophthalmol 74: 1066 - 1074, 1972.
7. Holt WS, Regan CDJ, Trempe C:- Acute posterior multifocal placoid pigment epitheliopathy. Am J Ophthalmol 81: 403 - 412, 1976.

BLOCH SULZBERGER SYNDROME

Qazi Masood MD; Iffat Hassan MD; Imran Majid MD.

***ABSTRACT**: A case of Bloch Sulzberger syndrome with involvement of CNS and eyes is described here in view of its rarity.*

***KEY WORDS**: Incontinentia Pigmenti, Bloch-Sulzberger syndrome*

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INTRODUCTION

Incontinentia Pigmenti, also known as Bloch-Sulzberger syndrome, is a hereditary disease transmitted as an X-linked dominant trait [1,2]. The disorder is thought to be lethal in affected males, a few surviving males believed to represent a spontaneous mutation consistent with the half chromatid model [2]. Thus more than 95% of the reported cases of this disorder in the world literature have been females. The characteristic features of this disorder include linear, splash like skin lesions of different types with variable involvement of the eyes, CNS and skeletal systems [1,3,4]. The skin lesions usually start at the time of birth or within the first few weeks of life and are vesicular to start with, arranged in a linear whorled or splash like pattern. Veruccous or lichenoid lesions usually follow these lesions in the second stage that occurs around the age of four to eight weeks. This second stage is seen in only one-third of patients and the sites involved are those affected by the initial vesicular lesions especially the dorsa of hands and feet affect those. In the third and final stage of cutaneous involvement gray or brown hyperpigmented streaks, whorls or even stellate macules are seen and these may involve sites not previously affected by the vesicular or lichenoid eruption [1,3,4]. Sometimes the pigmented lesions can be seen at the time of birth only suggesting an earlier intrauterine vesicular or inflammatory phase.

Extracutaneous manifestations occur in more than one third patients and include eye anomalies, dental abnormalities, CNS involvement and also skeletal anomalies in rare cases. The ocular lesions, seen in 30% of patients, include cataracts, uveitis, chorioretinitis, glaucoma, microphthalmos, retinal pigment mottling or telangiectasia, optic atrophy, nystagmus and strabismus. However the most typical abnormality of the eyes seen in this disorder is a retrolental mass with detachment of a dysplastic retina and this abnormality has been reported in 12% of patients[5]. Dental abnormalities occur in more than 60% patients and the lesions commonly observed are a delayed eruption, pegged or conical teeth and oligodontia. CNS involvement which occurs in 25-30% of patients is seen in the form of mental retardation, convulsions, spastic and lax paralysis, microcephalus and motor retardation. Skeletal anomalies are rarely associated and these include hemi vertebrae, extra ribs, cleft lip and palate etc.

CASE REPORT

A three-year-old female patient born from a non-consanguineous marriage presented to our department with the complaints of general pruritus with linear hyperpigmented lesions on the trunk and limbs. The hyperpigmented lesions had been there since the age of three months and had shown a mild regression only over the years. The pruritus was of two months duration and had shown little response to oral antihistamines. On questioning the parents gave a history of a vesiculobullous eruption on the third day of the child's life. The eruption had involved the trunk and lower limbs only in a classical linear pattern. The eruption had subsided within about two weeks without causing any significant problem to the child. There was no history of any verrucous or lichenoid lesions in the past. A few weeks after the initial eruption had subsided the parents had noticed linear hyperpigmented lesions at the same sites originally involved by the vesicular lesions. The lesions had progressed for sometime but then had remained almost stationary over the years.

There was also a history of generalized tonic clonic convulsions since the age of six to seven months for which the patient had been put on Valproic acid. The patient also had a history of delayed developmental milestones with mental retardation and the patient was unable to talk

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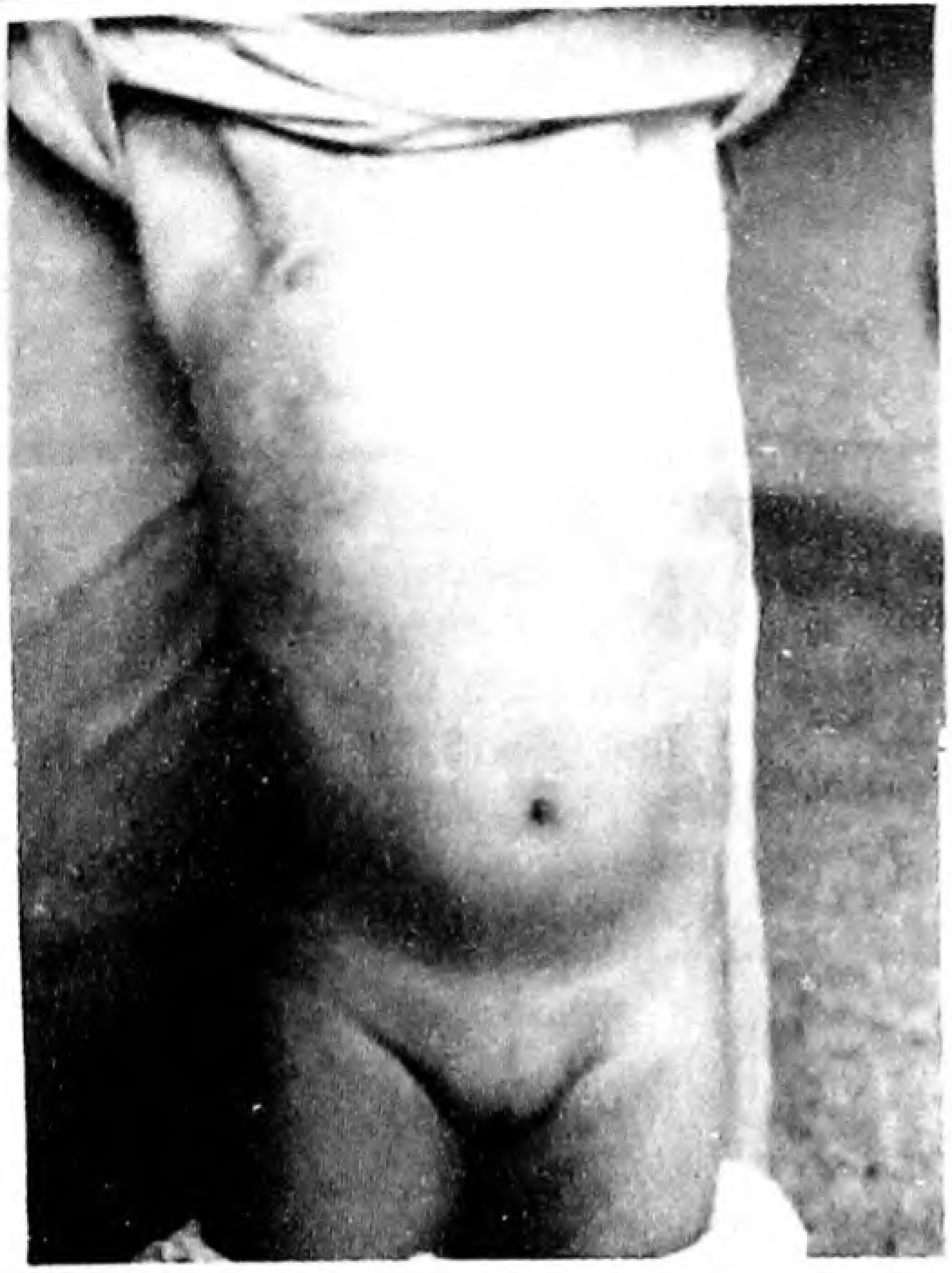
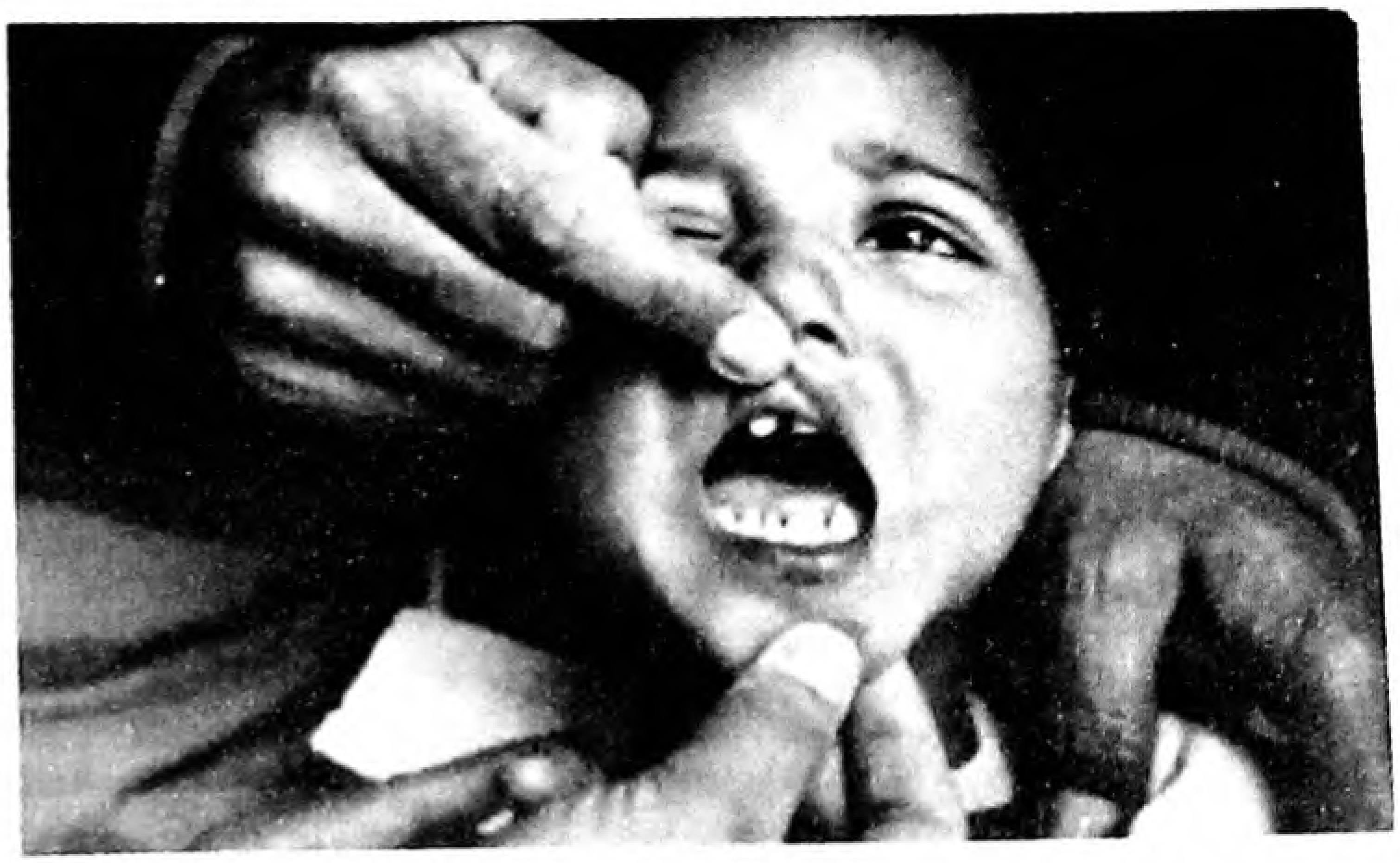
meaningfully even at three years of age.

On examination, the patient was of normal height and weight with no evidence of any physical retardation. Cutaneous examination revealed a whorl like, streaky macular hyperpigmentation involving the trunk and lower limbs and following no particular anatomic or neural distribution [Fig 1]. A generalized xerosis of the skin with follicular hyperkeratosis was also evident explaining the history of pruritus in the patient. No other evidence of an atopic diathesis was present in the patient. Nails, hair and mucous membranes were normal but the teeth were malformed and peg shaped especially on the lower jaw [Fig 2]. There was also a history of delayed dentition in the patient. Examination of the CNS revealed a moderate degree of mental retardation and spastic paresis of the left lower limb. Motor and sensory evaluation of the other three limbs revealed no abnormal findings. Ophthalmologic examination revealed no abnormalities except for an alternating divergent squint. Examination of the fundus was within normal limits and the skeletal system also revealed no abnormal features.



Specific laboratory investigations performed included a skin biopsy of the hyperpigmented macular lesions for routine histopathology, a complete slit lamp examination of the eyes and CT scan of the head and neck regions. The histopathology of the skin showed a normal epidermis with a lot of melanophages in the upper dermis. Rest of the laboratory tests were within normal limits.



The patient had a younger male sibling who was clinically normal as were both the parents. The female sex, presence of the typical skin lesions and the associated anomalies allowed us to make a diagnosis of Incontinentia Pigmenti.

References:

1. Wiklund DA, Weston WL. Incontinentia pigmenti. *Arch Dermatol* 1980; 116: 701-03
2. Shotts N, Emedy AEH. Bloch-Sulzberger syndrome [Incontinentia pigmenti]. *J Med Genet* 1966; 3: 1148-52.
3. Stawiski M, Van Goor K, Sturman S. Incontinentia pigmenti: a case report. *Int J Dermatol* 1981;20: 456
4. Carney RG, Jr. Incontinentia pigmenti. *Arch Dermatol* 1976; 112: 535-42
5. Carney RG. Incontinentia pigmenti. A world statistical analysis. *Arch Dermatol* 1976; 112: 535.

RENOCOLIC FISTULA SECONDARY TO RENAL CALCULUS WITH TUBERCLUOSIS

I. Shah M.S.,F.I.C.S.; Rahul Gupta M.S.; C.L. Gupta M.S.,M.C.H.; Irfan Robani M.D.

Abstract: *Renocolic fistula is very rare. Till now 100 cases have been reported in world literature[1]. We report an unsual case of Nephrocolonic fistula caused by a renal calculus with Genitourinary Tuberculosis. Patient was successfullly managed by single stage surgery (Nephrouretrctomy, Fistulectomy and primary repair of the colon) with anti tubercular drugs.*

Key words: *Fistula, Colonic, Tuberculosis, Stone.*

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Introduction

The occurrence of Genito urinary tuberculosis has ot yet considerably decreased in the developing countries[2] in spite of effective anti-tubercular drugs. Renocolic fistula with renal calculus and genitourinary tuberculosis is a rare entity. The incidence has greatly fallen in recent decades because of advances in diagnosis[3] and treatment of renal disease such as tuberculosis. No organ is resistant to tuberculosis and world wide genitourinary form of disease accounts for 14%. We report a case, patient with renocolic fistula secondary to renal calculus with tuberculosis. Patient was diagnosed by cystopanedoscopy, right retro-grade pyelography and was managed by surgery.

Case Report

Thirty years female patient was admitted with right lumbar pain and recurrent urinary tract infection, with frequency and dysuria and constipation for last six months. On physical examination her respiratory and cardiac status was normal. Urine microscopy revealed pyuria and E. Coli was grown on culture. Her haemoglobin was 9.6gms with raised pollymorphs. Renal parameters were within normal limits. Ultrasound of abdomen and pelvis revealed right renal calculus with back pressure chages. Patient was subjected to intravenous pyelography which revealed right renal calculus with non-functioning right kidney. Patient underwent Cystopanendoscopy with right retrograde pylography under local anesthesia which showed patulous pulled up right uretric orifice with hyperemia, and retograde showed normal right ureter and stone in the right pelvis with dialated calyces and dye leaking into the right asending colon (fig). Diagnosis of right renal calculus with renocoloic fistula was made. Patient was put to sleep with nitrousoxide and haloothane and was explored via trans peritoneal approach. Tract between asending colon and right kidney was established. Right nephrouretrectomy with excision of fisutlous tract and primary closure of colonic fustula was done. Pathological examination of the specimen revealed right kidney containing staghorn calculus with epitheloid granlomas with nenrosis and langerhans cells suggestive of tuberculosis. Patient had uneventful post operative period and was put on four drug regieme i.e. INH, Refampicin, Ethembutol and Pyriznamide for three months followed by three drug regiem for next three months. Patint had excellent recovery annd repeat cystoscopy showed normal bladder mucosa.

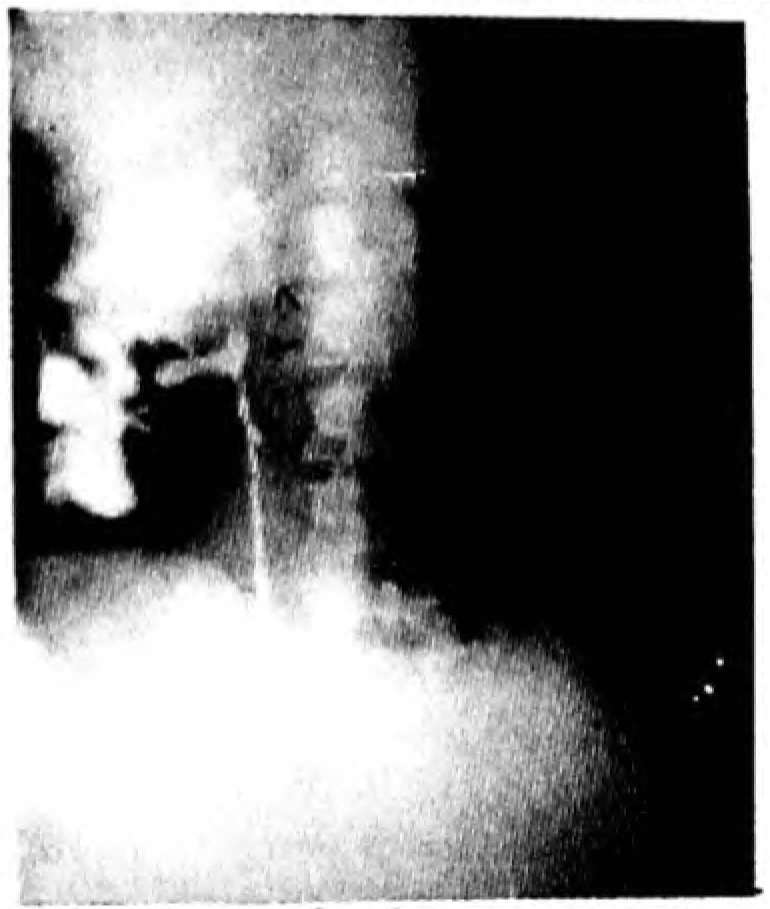

Fig. 1 Retrograde pyleography showing renocolonic fistula with staghorn calculus in pelvis

Discussion

Spontaneous renal fistulisation to neghboring viscera is not uncommon[4] however spontaneous fistulisation to colon

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without previous renal surgery is very rare. Although the first recorded case was described by Hippo crates and till date approximately 100 cases have been reported in literature. Fistula between the upper urinary tract and the bowel usually represent progression of renal disease[4]. The underlying cause for such fistulas are[1] chronic inflammation without stone disease (e.g. tuberculosis, bacterial and non bacterial) or with stone (usually staghorn) and obstruction (stone, stricture of ureter, pelvic kidney), trauma to gastrointestinal in origin. In our case Nephrocolonic fistula most likely progressed as chronic infection (tuberculosis) renal stone Pyonephrosis and abscess formation casused peripylititis and perinephritis and adhesions of the renal pelvis to underlying colonic area and led to subsequent development of a colorenal fistula. The diagnosis is made radiologically which include intravenouspylography and retrograde pyelography as done in our case. C.T. scan administration of methoylene blueorally or indigocarmine intravenously which is excreted with urine or stains the feaces is sometimes used. Some times preoperative investigations may be in sufficient and in such cases the diagnosis is made intraoperatively.

The treatment of renocolic fistula depends on the uderlying etiology and the degree of renal function. Now with the newer anti tubercular drugs and more surgical facilities available major surgery can be accomplished with a cover of anti tubercular drugs for a short span with primary repair of colonic fistula and nephrourerectomy can be performed safely.

References:

1. Renocolic fistula secondary to chronic pyelonephritis. M. Yildiz, A. Atan, LL. Aydoganli, T. cengiz. Zakalin. Int, Urology and nephrology 1993;(25)3,229-233.
2. Primary repair of colorenocutanous fistula in patients of Genitourinary tubercluosis A.K. Hemal, N.P. Gupta, S.N. Wadhwa, M. C. Songra, U.N. Bhyan. Urol Int 1994;52:41-44.
3. Cutanous nephrocolic Fistula as a consequence of a kidney stone. Karlijn J.P, Van Wessem, Nichh. J. van. Southeren Medical Journal Sept. 2000;(93)9:934.
4. Fistulas of the genitourinary tract An atlas and textbook oof urologic imaging Lang EK, Fritzche p. Philadelphia; Sanders, 1990:2579-2583.
5. Renocolic fistula demonstrated by antegrade pyelography. Newman JH, Jeans WD. Br J Uro, 972;44:692-697.
6. Reno-colic fistula. Mander BJ, Menzies D, Moson RW. JR Soc Med 1993;86:60-602.
7. Reno-alimentary fistula: an unusual urological problem. Bissa-da NK. Cole AT, Fried FA. Urol 1973;10:273-276.

PLASMODIUM VIVAX MALARIA PRESENTING WITH HEMOLYSIS

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Plasmodium vivax malaria is common in Indian subcontinent (1) but it rarely produces hemolysis (2). This is because peripheral sequestration does not occur in the vivax malaria. Although the magnitude of parasitemia is limited by the requirement for young red blood cells (reticulocytes) in P.vivax infection the hemolysis associated may stimulate haematopoiesis enough to increase the number of reticulocytes and may thus permit parasitemias subsequently greater than 1 to 2%(3). The unusual presentation of P. vivax malaria presenting with hemolysis is presented.

Sixteen-year boy from Uri, Northern outskirts of Kashmir valley was admitted with high grade intermittent fever (102-104°F) associated with chills and rigors and exertional breathlessness of one month duration without any history of bleeding from any site, abdominal or chest pain, joint swelling, rash, headache, cough and bone pains. The clinical examination revealed a thin built person with pallor, tachycardia, mild icterus and moderate splenomegaly. Investigations showed haemoglobin 5.4 gms/dl with normal other cell series. Peripheral blood film (PBF) showed microcytes, moderate anisopoikilocytosis with tropozoites and merozoites of P.vivax. PBF examination at 8 hour intervals for consecutive 3 days was done to rule out double infection with P.faciparum, Hemolytic profile revealed reticulocyte count of 7%, Sr. bilirubin (2.8 mg/dl, indirect) LDH 829 mg/dl, plasma haemoglobin 20 mg/dl with normal G6PD, Osmotic fragility and foetal Hb, a negative coombs test (direct and indirect) and Sucrose lysis test. Three blood cultures after 48 hours incubation were sterile. Dip stick antigen capsular assay for P.falciparum, widal and brucella agglutination tests were negative. Bone marrow aspiration showed hypercellular erythropoiesis, normal granulopoiesis with mild increase in eosinophils and adequate iron stores with sterile bone marrow culture incubated for 48 hours. No malarial parasite was seen in the bone marrow aspiration. Radiology including chest x-ray PA view, ultrasound abdomen and echocardiography were normal.Upper GI endoscopy was also normal. He received chloroquine 600 mg stat, followed by 300 mg at 6 hours, 24 hours and 48 hours and became afebrile after 24 hours and his spleen decreased in size in 7 days. Serum bilirubin came to normal after 14 days. Primaquine tablet 7.5 mg twice a day was prescribed for 14 days. After 6 weeks the patient was asymptomatic with no clinical evidence of anaemia, jaundice or splenomegaly. His repeat haemoglobin was 14 gms/dl and reticulocyte count was 1.5 Cnm, serum bilibrubin was 1 mg/dl and plasma Hb was 6 mg/dl. PBF for malaria parasite done at 6 different times in 3 days was negative.

Malaria is uncommon in Kashmir, but it is seen in the outskirts of the valley. Actually ill patients with malaria typically have either falciparum or P.vivax infection, although 5 to 7% of infections are due to more than one species (3) P.vivax infection clears with chloroquine and chloroquine resistance to P.vivax is extremely rare after a proper treatment. Chloroquine resistant P.falciparum continues to multiply and causes symptom (3).

Our patient became a febrile after 24 hours of treatment and blood smear done twice a day for 3 days after 72 hours of treatment were negative. Plasmodium falciparum infection was excluded by repeated PBF examinations and capsular antigen test.

REFERENCES

1. Nicholas J White, Joel G.Breman. Malaria- Harrisons Principles of Internal Medicine 2001, 15th ed. Vol.1, 1203-12
2. Dacie JV et al. Secondary or symptomatic haemolytic anaemia. The haemolytic anaemia. Grene and Strathen New York, 1967, Part III Chapter 15, page 908.
3. Shashank R Joshi. Malaria- current status and challenges. Pharmacology for general practioners June July 2000, Vol 2, no. 1,17-31.

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ELECTROCARDIOGRAPHIC CHANGES IN HYPOKALEMIA-A RARE PRESENTATION.

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A 60 year old female patient presented to the emergency room with one week history of epigastric pain, recurrent vomiting and worsening weakness. She had history of recurrent hypokalemic weakness of ten years and records showed equivocal etiology of hypokalemia. Physical examination revealed mild to moderate dehydration with B.P 90/60 and muscle power of grade 2-3. Respiratury .rate was normal, however single breath count was reduced. Relevant laboratory findings included prerenal azotemia, plasma K 1.7meq/1, plasma Na 132meq/1 and metabolic alkalosis. EKG showed PR prolongation, wide QRS, marked ST segment depression, prominent U waves, very low voltage T waves, RBBB, left posterior hemiblock and NW axis (marked right axis deviation). After ruling out acute coronary syndrome patient was managed for acute gastritis and hypokalemia. Serial monitoring of plasma K and EKG showed progressive disappearance of abnormal EKG findings with rise in plasma K.

The changes in EKG correlate with plasma K fairly well, being found in 78% of patients with plasma K levels below 2.7meq/1, in 35% of those with K levels between 2.7-3.0 meq/l, and in 10% with plasma K between 3 and 3.5 meq/l.

The abnormal and delayed repolarisation that occurs in hypokalemia is best expressed as QU, rather than QT, prolongation. As plasma K falls, the ST segment becomes progressively more depressed and there is gradual blending of T wave into what appears to be a tall U wave. When hypokalemia is severe, the QRS complex may widen slightly, in a defuse manner. The P wave and QRS amplitude may be increased and PR interval is often prolonged. Hypokalemia promotes the appearance of supraventricular and ventricular ectopic rhythms, being enhanced by increased automaticity and / or facilitation of reentry. Bundle branch and' fascicular blocks have not been reported so far in hypokalemia, although they can occur in hyperkalemia due to depression of intraventricular conduction by high plasma K.

In this report, RBBB and LPHB, were abnormal EKG findings as manifestations of hypokalemia. This warrants further studies to know incidence and mechanism of bundle branch and fascicular blocks in hypokalemia.

References:

1. Castellanos, A. and others, Hurst's The Heart 8'" edition, The resting electrnca."dingram,321-356.
2. Rardon, D; F and Fisch, C., Electrolytes and the Heart, Hurst's The Heart,8'" edition, ch.37,759-774.
3. Surawicz B. (1963) Electrolytes and the electrocardiogram. Am. J.Cardio1.12,656
4. Surwicz B. (1976) Relationship between electrocardiogram and electrolytes Am. Heart J.73,814

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ACUTE ABDOMEN FOLLOWING TORSION OF OVARIAN CYST

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A case of. ovarian cyst with torsion managed in a community health centre is reported. A 29 years old Muslim female GSP3A1 was admitted with complaints of amenorrhoea 2 years, pain abdomen 16 hours and vomiting 10 hours duration. Examination revealed ill looking female with features of tachypnoea, anemia, malnutrition, tachycardia, sweating and hypotension. Whole abdomen was distended, skin stretched, umbilicus everted, and restricted respiratory movements. Abdomen was tender all over. She had an intra-abdominal mass involving the whole abdomen and only the upper margin of the mass could be felt in the region of epigastrium. Percussion note was dull all around, shifting dullness and fluid thrill could not be elicited. Clinical examination and x-ray abdomen did not show any evidence of fetus. A provisional diagnosis of acute abdomen (torsion ovarian, mesenteric mass) was made and patient operated under spinal anaesthesia by midline incision. A huge right ovarian cyst involving the whole abdomen with only a twist at the pedicle and gut pushed to epigastrium and splenic region was seen. All measures to deliver the cyst failed and cyst was delivered only after aspirating 3300ml of cystic fluid then right ovarian cystectomy with salpingo-oophorectomy was done. The cyst still weighed 12.7 kg (Fig. 1). The patient had uneventful postoperative period. She underwent LSCS (Lower segment caeserean section) in the same hospital 20 months later, when she presented with full term pregnancy with labour pain with bad transverse lie.

The case highlights the myth and beliefs which the people still have. She was denied medical consultation on the pretext that she is conceiving but because of her sins/and angry goddess around her she could not deliver. Also it was believed that instead of a baby she has some ghost inside.

It was only after she got severe pain abdomen and vomiting that the villagers decided to shift her to a hospital with the consideration that these ailments can be taken care of by the doctors.

We hereby impress that after all the mesures of referral to good hospital fail whatever in&astructure is available, every attempt should be made to operate such patient to save the precious life least they can not reach to distt. hospitals not to speak of medical college hospitals/ institutes of the state which are far away from such remote centres.

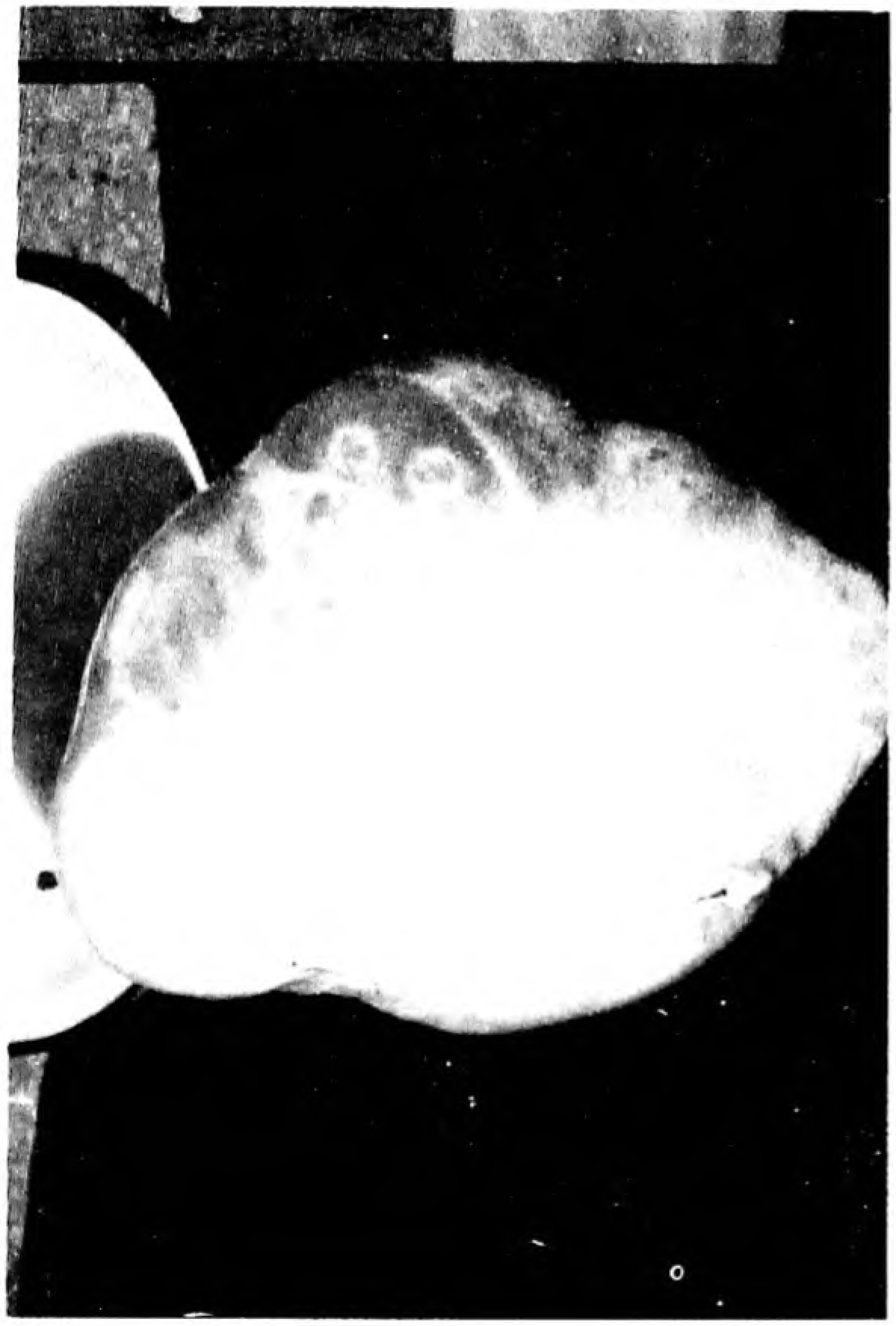

Fig. 1 Resected Ovarian Cyst which had undergone torsion

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FIBROID UTERUS — DELIVERED PER VAGINUM

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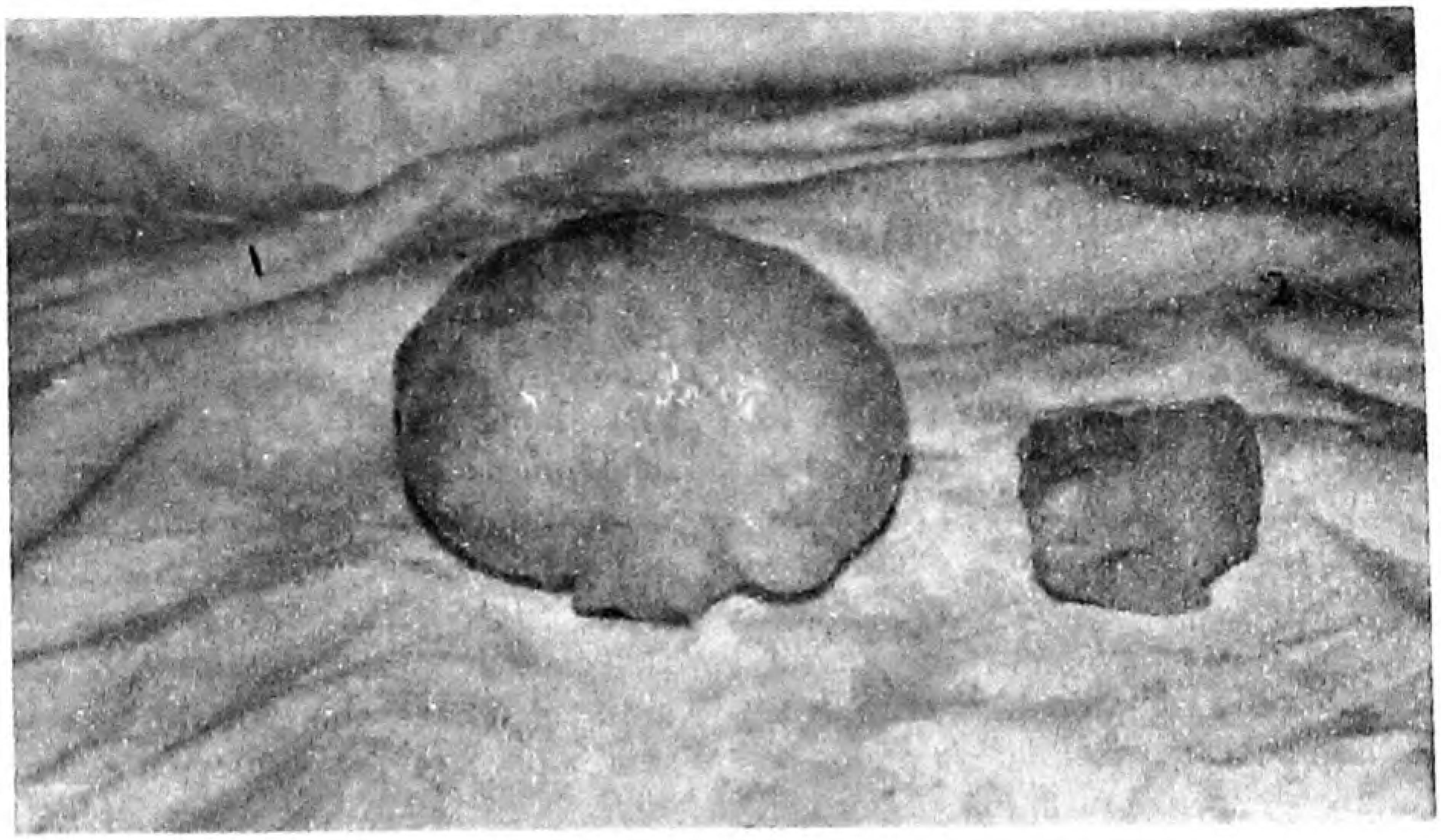

Fig. 1 : Fibroid uterus delivered per vaginum
i) Left Bigger One Fibroid Uterus.
ii) Right Sammler One Exicised Uterus.

A 44 year old female was admitted with complaints of excessive bleeding per vaginum, easy fatiguability, breathlessness of 12 years duration. She had married 26 years back but had never conceived instead had history of Dysfunctional Uterine bleeding from last 20 years. Examination revealed ill looking bed ridden tachypnoeic patient. She had severe anaemia and was emaciated. An intra-abdominal mass involving hypogastrium right and left iliac fossa and umbilical region was observed. Per vaginum examination revealed blood stained vulva, blood ooze per vaginum and a smooth surface mass involving the whole vagina. Fingers could not be negotiated around mass up into pelvis, it bled on touch, was very huge and encroaching on all the corners. Investigation revealed Hb 4.2 gm, platelet 1.4 lakh, urea 75, creatinine 1.4, and sugar 90.

Laparotomy by midline incision was done. Uterus mobilized and hysterectomy done. After doing hysterectomy mass was seen rising from the fundus of uterus by a short pedicle but the mass could not be delivered per abdomen. More than fifty percent of mass was below pelvic brim. A formal episiotomy was done and vaginal forceps used to extract out the mass. It weighed 6.3 kg (Fig.1). Histopathological report was consistent with fibroid uterus. She had uneventful postoperative recovery and was doing well at 4 years of follow up. Though laparotomy with ligation of feeding vessel to the tumour was the plan. On table whole tumor was removed and patient was given a cure.

We emphasize emergent surgical intervention even in community health centre and especially if the patient and attendants are reluctant in taking or shifting the patient to specialised centre.

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MECHANICAL VENTILATION IN ACUTE RESPIRATORY DISTRESS SYNDROME

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Acute respiratory distress syndrome (ARDS) as per American - European concensus conference is defined as acute onset respiratory distress with PaO2/FiO2 ≤ 200 regardless of PEEP, chest X-ray showing bilateral alveolar or interstitial infiltrates consistent with pulmonary edema and no clinical evidence of left atrial hypertension or PAOP ≤ 18 mmHg if pulmonary artery catheter is in place. The most widely practiced method for quantifying ALI/ARDS is Murray Scoring system that includes the number of quadrants consolidated on chest X-ray, hypoxemia score (PaO2/FiO2) and if ventilated compliance and PEEP also.

The therapeutic goals in ARDS management are treatment of underlying event, cardiorespiratory support and specific treatment targeted at lung injury. The evolving principles of mechanically ventilating injured lungs originate from the fact that there are three distinct populations of alveoli in ARDS - units with normal inflation properties, consolidated units that are uninflatable and units that are atelectatic but potentially recruitable. The last two populations of alveoli occupy mostly dependent regions of the lungs suggesting thereby heterogenous involvement.

Traditional mechanical ventilation strategy for ALI/ ARDS;

Ashbaugh in 1967 first reported the beneficial physiological response to PEEP and shortly thereafter the traditional strategy for the use of mechanical ventilation (MV) in ALI/ARDS management was developed and remained unchanged until few years ago. It can be summarized as follows :

- i) Volume cycled ventilation using tidal volume(VT) of 10 to 15ml/kg
- ii) respiratory rate of maintain a normal pH and
- iii) PEEP adjusted with respect to PaO2 and FiO2.

The goal of this strategy was to achieve oxyhaemoglobin saturation (SaO_2) of $\geq 90\%$ at nontoxic FiO2 i.e. ≤ 0.6. As the imaging technology improved over years radiographic barotrauma (interstitial emphysema, gas cysts,intraparenchmal tension cavities, bronchopulmonary damage and systemic gas embolism) became increasingly recognized, this strategy faced many challenges and hence underwent modifications. In 1970s intermittent mandatory ventilation (IMV) was introduced in ALI/ARDS management and was the most commonly used ventilatory mode and some investigators used PEEP$\geq$50 cm H_2O.

NEW MECHANICAL VENTILATION STRATEGY FOR ALI / ARDS :

In mid 1980s animal experiments convincingly demonstrated that increased tidal volume and increased plateau and peak pressures initiated or propagated acute lung injury (ALI). This observation was supported by C.T. scan images in ARDS patients which consistently demonstrated patchy rather than diffuse homogenous lung injury. Since much of the lungs in ARDS are edematous, atelectatic or fibrotic this exposes some relatively normal units to increased regional tidal volume (VT). This ventilatory strategy is directed at the mechanisms of lung injury in ARDS including high stretch and cyclic reopening of atelectatic alveoli. Its components are:

- i) Use pressure - targeted ventilation (VC or PC mode) limiting VT to 4-8 ml/kg and plateau pressure <35 cm H2O.
- ii) Allow $PaCO_2$ to increase if necessary to meet the above objectives. The resulting increase in $PaCO_2$ though not desired is a necessary by- product of this strategy i.e. permissive hypercapnia.
- iii) Adjust PEEP for PaO_2/FiO_2 and heep PEEP above lower inflection point of pressure volume curve.

Guidelines for bedside management :

1. Avoid alveolar over- distension
2. Maintain $FiO_2 < 0.6$
3. Use sufficient PEEP to prevent tidal recruitment - derecruitment.
4. Mode of ventilation is less important than goals 1 to 3
5. Tolerate hypercapnia if necessary to achieve goals 1 to 3.

These goals can be achieved in more than 90% of newly ventilated ARDS patients and in the remaining cases one may have to reset to salvage therapies. On presentation give oxygen by high flow mask but one rarely achieves a tracheal $FiO_2 > 0.6$ by this. Intubation should be performed early and electively (when you anticipate that MV will be

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required) without waiting for severe respiratory failure to supervene. Role of non-invasive positive pressure ventilation (NIPPV) is not established in ARDS management because of associated haemodynamic instability, multi-organ dysfunction, altered sensorium and longer course. Sedatives and muscle relaxants should be used liberally in ventilated ARDS patients (especially while using PCV) as these agents decrease O_2 consumption and CO_2 production by skeletal muscles and also help in patient - ventilator synchrony.

Initial ventilator settings should be FiO_2 1.0, respiratory rate 20-30/mt and VT 6-8 ml/kg using either ACMV or SIMV mode and if one chooses PCV, and inspiratory pressure of 30 - 35 cms. Once SaO_2 is >90% first priority is to decrease FiO_2 to <0.6 to minimize further lung injury. Low saturation in addition to anaerobic metabolism can lead to cardiac problems like acute DCM, global hypokinesia and arrest.

Positive End Expiratory Pressure (PEEP):

PEEP is used to recruit collapsed alveoli (prevention of derecruitment at end expiration), diminish intrapulmonary shunt, permit reduction in FiO_2 and to translocate fluid from flooded alveoli to interstitial space. This results in increase in compliance due to increase in FRC and hence improved gas exchange. It is important to keep airway disconnections to minimum since decrease in PEEP even for short periods leads to alveolar derecruitment and rapid haemoglobin desaturation. If patient fails to respond to PEEP one should suspect focal lung processes like lobar pneumonia, atelectasis etc. Approach to PEEP in ARDS in still evolving and currently two strategies are being practiced in clinical medicine.

1. **Least PEEP approach**
   In this strategy a PEEP of 15-20 cm H_2O is applied in a single step using a VT of 6-8 ml/kg.However high plateau pressure (45-50cms) and hypotension may be encountered by this. Hypotension is managed by decreasing PEEP temporarily and intravenous fluids and inotropes. Once $FiO_2 \leq 0.6$, PEEP is reduced by 2.5 cm H_2O with decrements at least 15 minutes apart seeking the least PEEP that gives $SaO_{2>}$ 90% . In some patients PEEP must be increased to more than 15-20 cm H_2O to get FiO2 < 0.6. In these patients one may use ancillary strategies like packed RBC transfusion, cardiac output augmentation by dobutamine and decrease in O_2 consumption by sedatives, paralysis, cooling, etc or resort to salvage ventilatory options.
2. **Open lung approach:**
   This strategy uses PEEP not just to avoid O_2 toxicity but also as a lung protective mechanism. Here pressure - volume (PV) curve is constructed by increasing VT from 100 to 1000 ml noting pressures after an inspiratory pause - all with zero PEEP. It takes approximately 30 minutes. LIP of PV curve is determined and then PEEP is set at 2 cm higher. If it is impractical to determine PV curve daily then PEEP is set at a typical value required to exceed LIP i.e. 17 cm H_2O (Amato trial). As oxygenation improves over days PEEP is reduced and mode is changed from PCV to PSV. This approach provides no guide to PEEP reduction.

Once PEEP is adjusted and FiO_2 decreased to <.6 - mode, respiratory rate and VT should be reevaluated. There is no clear benefit to one mode or another. PCV is simpler to use since it does not require repeated determination of plateau pressures however equivalent benefit can be provided by the more preferred modes like ACMV or SMV. Inverse ratio ventilation (IRV) can be included with any of these modes.

Innovative therapies:

1. **Permissive hypercapnia :**
   It is the simplest approach to decrease ventilatory workload, hazards of high pressures and total number of machine cycles needed per minute. It is chosen to avoid injurious airway pressures than to maintain normal ventilation. It has a clear benefit in terms of compliance and gas exchange in lung - protective ventilatory strategy.

   It requires deep sedation and paralysis. Gradual increase in $PaCO_2$ is accompanied by small shifts in intracellular pH and is generally well tolerated even to very high levels. It is recommended that $PaCO_2$ should be allowed to increase at 10 mmHg/hr. Permissive hypercapnia decreases mortality not only in ARDS but also in status asthmaticus. When ARDS patients are ventilated with volume or pressure limited ventilation $PaCO_2$ typically rises to 60-70mmHg and pH drops to 7.2 but in some cases $PaCO_2$ may rise to >150 mmHg and pH may drop to <7.0. Healthy human volunteers have tolerated $PaCO_2$ of 250 and pH of 6.72 without serious consequences because our body has efficient cellular compensatory mechanisms.

   Adverse physiological effects of respiratory acidosis include cerebral vasodilation ('^ ICP), coronary vasodilatation (leading to steal - syndrome), systemic vasodilatation ("v BP), decreased myocardial contractility, cellular metabolic dysfunction and pulmonary and renal vasoconstriction. From this it is obvious that permissive hypercapnia should not be used in ARDS associated with increased intracranial pressure, myocardial ischemia, hypotension and/or LV failure on inotropes, pulmonary hypertension with acute RV failure and severe metabolic acidosis.

2. **Partial liquid ventilation (PLV):**

   It is an exciting new approach to recruit collapsed lung units by filling these with a gas exchanging liquid perfluorocarbon (PFC). These PFCs are biologically and chemically inert (non-histotoxic), free of toxic metabolites, clear but radiopaque, have no significant systemic absorption and are eliminated from lungs by evaporation in hours to days. These liquids partly or completely fill to FRC and thereby function as PEEP. By virtue of hydraulic column PFCs are able to recruit most dependent alveoli which PEEP is not. These liquids dissolve very high volumes of O_2 and CO_2, distribute easily throughout the lungs and gravitate to the regions at highest risk for airway closure and alveolar collapse. PFC filled alveoli are theoretically supposed to be protected against lung infection and damage.

   The most extensively studied PFC is perfloro-octyl-bromide (Perflubron) which has density twice that of water and therefore gravitates to the most dependent regions and it also reduces the interfacial tension that normally exists between air and surfactant depleted alveoli. Unlike endogenous or exogenous surfactants, surface tension lowering effect of perflubron is not inhibited by proteinaceous exudates. Perflurbron may find application as lavage fluid in place of normal saline as it is more effective in preserving gas exchange and removal of alveolar debri.

   PFCs impair imaging assessment because of their extreme radiodensity and thus it is difficult to use chest X-ray for detection of infection and follow progress. The side effects of PFCs include pneumothorax and mucous - plug formation. Their final place in ARDs management is not clear as yet.

3. **Prone Positioning:**

   It is a relatively safe and underutilized technique that produces significant improvement in gas exchange in ARDS. Benefits are observed in minutes to hours and then wane off after several hours and some responders continue to maintain better gas exchange even when turned back to supine position. It improves gas exchange in 50-70% patients treated in early phase of ARDS allowing reduction in both FiO_2 and PEEP. Various mechanisms responsible for better gas exchange in prone position include recruitment of dorsal lung units with improved V/Q matching, increased FRC, change in regional diaphragm motion, better secretion clearance and enhanced lymphatic flow.

   The frequency and duration in supine and prone positions is titrated to gas exchange response, however, most experienced centers turn patients once or twice daily with equal time in each position. The potential problems of prone positioning include displacement of endotracheal tube (ETT), and central venous lines, difficulty in nursing care and pressure necrosis of nose, face and ears. In addition there is always a threat of desaturation, arrythmias and hypotension, however these are usually transitory and can be minimized by prior sedation, suctioning and 100% oxygen.

4. **Tracheal gas Insufflation (TGI):**

   It is a useful adjunctive technique that involves introduction of fresh gas near carina via a modified ETT. This added flow washes out CO_2 rich gas out of trachea and through turbulance out of small airways as well thereby decreasing the anatomical dead space. In general in ARDS, TGI with 100% humidified O_2 at4 UL/mt decreases $PaCO_2$ from 108 TO 84 mmHg. The potential risks associated with TGI include tracheal erosion, O_2 toxicity related to unknown FiO_2 and haemodynamic compromise or barotrauma due to occult auto-PEEP.

5. **High frequency ventilation (HFV):**

   High frequency jet ventilation (HFJV) employs respiratory rate of 60-300 /mt and VT of 1-5 ml/kg. Mechanism of gas exchange is poorly understood, two possible hypothesis include axial diffusion and bulk flow. Multiple trials of HFV have failed to show its superiority over conventional ventilation (CV) in terms of improved oxygenation, reduced barotrauma and decreased days of MV. Currently it is regarded as a salvage therapy for ARDS refractory to CV.

6. **Inverse ratio ventilation (IRV):**

   It involves use of prolonged inspiratory time (1:E>1) with either volume cycled or pressure cycled modes of MV. A subset of patients with hypoxaemia refractory to conventional modes respond to IRV but there is no way to prospectively identify these. The exact mechanism by which IRV improves gas exchange is not know however the two possibiltieis include increased alveolar recruitment at lower airway pressures and optimal distribution of ventilation. But there is no role of intrinsic- PEEP or gas traping in improving oxygenation.It is crucial to note that both auto-PEEP and higher mean alveolar pressure typical of IRV tend to decrease cardiaç output leading to hypotension. Inverse ratio - PCV is sometimes used for severe ARDS treatment. Presently IRV is considered only a salvage therapy for refractory ARDS.

7. **Extrapulmonary gas exchange :**

   Extracorporal membrane oxygenator (ECMO) adequately oxygenates the blood while allowing lungs to rest and recover. Extracorporeal CO_2 removal ($ECCO_2R$) is carried out by veno-venous techniques. Both appear to be attractive technologies but are not supported by clinical outcome studies and thus have little apparent future in ARDS management.

8. **Intravenacaval oxygenator (IVOX):**
It is a small membrane lung inserted into inferior vena cava (IVC) that is particularly beneficial in patients having hypoxaemia and hyper capnia. This device is capable of limited gas exchange thereby limitng its widespread use. IVOX has good future provided its gas exchange capacity increases.
ARDS has resisted pharmacological manipulations for almost 30 years however various drugs have been used with little success. Almitrine wich is administered i.v. increases mean PAP and cardiac output without any change in systemic BP. It increases respiratory drive and also improves V/Q matching. It lacks significant adverse effects but its effect is short lasting and PaO_2 returns to baseline soon after the drug is discontinued. Since surfactant in ARDS patients is structurally and functionally abnormal, artifical inhalational surfactant as a replacement therapy could be a promising modality in future. Though it produces dramatic therapeutic effects in neonatal respiratory distress syndrome, in ARDS it is as yet experimental only. The three selective pulmonary vasodilators that have been used in ARDS treatment include inhaled nitric oxide (NO), PGE_1 and aerosolized prostacyclin (PGI_2). Inhaled NO increases V/Q by decreasing intrapulmonary shunting producing an improvement in PaO_2 in ARDS in 50-70% patients, however, the effect beyond 72 hours is not sustained. Rapid inactivation of inhaled NO by Hb -binding prevents unwanted systemic side effects but also mandates continuous delivery to ventilatory circuit. If continuous delivery of NO is interrupted, precipitous and life threatening hypoxemia and RV failure can occur. These selective pulmonary vasodilators have not made any significant difference in ARDS severity, duration or mortality in controlled trials and are not recommended for routine management. Glucocorticoids have no proven benefit in exudative stage of ARDS except when the respiratory failure itself is caused by an inflammatory disease known to respond to steroids e.g. acute eosinophilic pneumonia. On the contrary steroids may increase infection rate, duration of MV and overall mortality. In fibroproliferative stage of ARDS steroid may have beneficial effect by virtue of their collagen synthesis inhibition. Ketokonazole an antifungal drug decreases the incidence and mortality of ARDS in critically ill surgical patients by blocking the synthesis of proinflammatory prostaglandins and leukotrienes.

Patient Monitoring:

Continuous pulse oximetry is reliable except in gross hypoperfusion. Arterial blood gas (ABG) measurement is frequently needed on first day and after major interventions or change in clinical parameters. Right heart catheterization is indicated when volume overload is suspected, otherwise it does not add to the overall outcome. If there is no improvement in 10-14 days of MV, tracheostomy should be considered. When FiO2 can be reduced to 0.4 to 0.5, PEEP should be decreased by 2-3 cms H_2O with several hours of observation between adjustments. When $FiO_2 \leq 0.4$ and PEEP <7.5 evaluate the patient for spontaneous ventilation (CPAP / T-piece) and thereafter extubation.

References:

1. Maggiore SM, Brochard L. Pressure-volume curve :methods and meaning Minerva Anestesiol 2001 Apr;67 (4):228-37
2. Cole FJ Jr , Shouse BA. Alternative modalities of ventilation in actue respiratory failure Surg Annu 1995;27:55-69
3. Hickling KF: Low volume ventilation with permissive hypercapnia in the adult respiratory distress syndrome. Clin Intensive Care 1992;67-78
4. Hickling KG: Ventilator management of ARDS : Can it affect the outcome? Intesive Care Med 1990;16:219-226
5. Gattinoni L, Pesenti A, Avalli L, et al: Pressure-volume curve of total respiratory system in acute respiratory failure.Computed tomographic study .Am Rev Respir Dis 1987;136:730-736
6. Hickling KG, Henderson SJ, Jackson R:Low mortality associated with low volume pressure limited ventilation with permissive hypercapnia in severe adult Respiratory distress syndrome. Intesive Care Med 1990;16:372-377
7. Tuchschmidt J, Fried J, Astioz M, et al : Elevation of cardiac output and oxygen delivery improves outcome in septic shock Chest 1992;102:216-220
8. Marini JJ, Kelsen SG: Retargeting ventilator objectives in adult respiratory distress syndrome.Am Rev Resir Dis 199;2146:2-3
9. Pesenti A: Target blood gases during ARDS ventilator management. Intensive Care Med 1990;16:119-321
10. Capeller G, Toth JL, Walker P, et al: hamodynamic effects of permissive hypercapnia, Abstr. Am Rev Respir Dis 1992 ;145: A527

EYE IN HEMATOLOGY (PART I)

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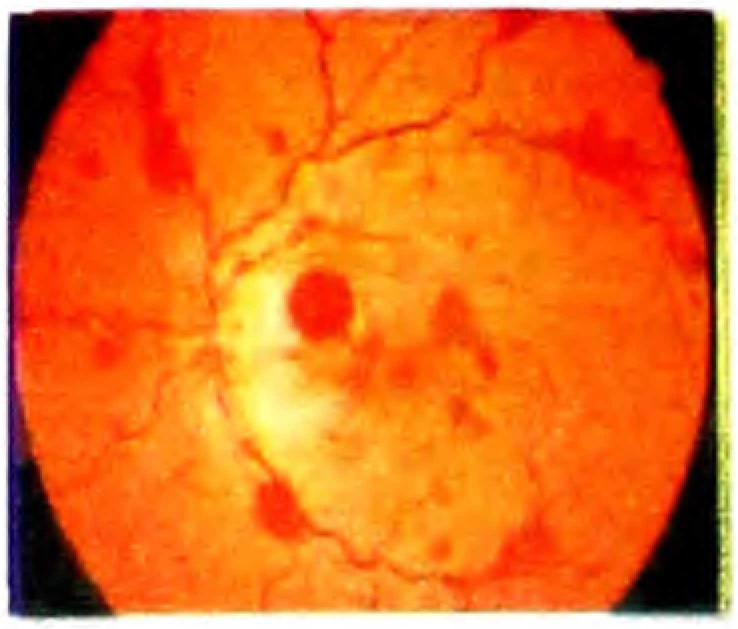
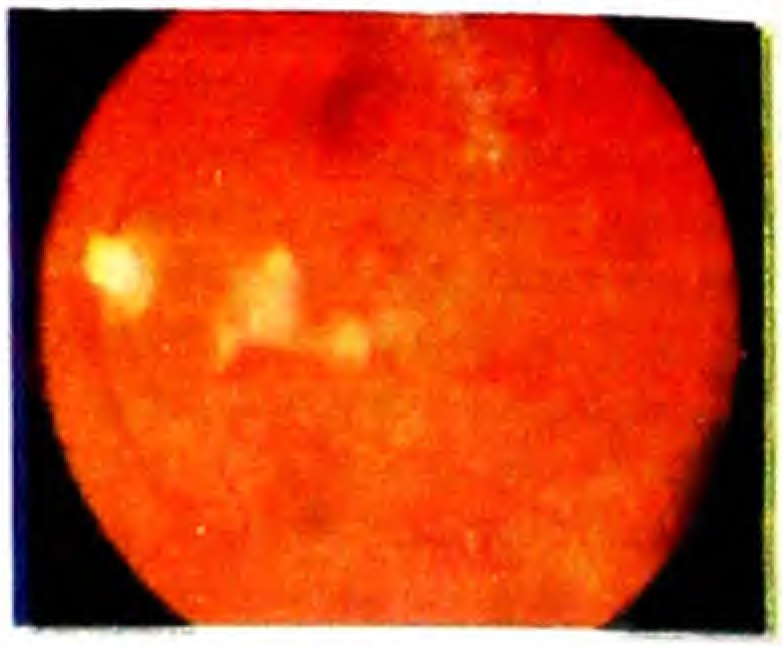
Introduction

- The unique vascular anatomy of the Eye makes the relationship between hematology and ophthalmology very close one.
- The ophthalmic clinician are the first witness of the various hematological disorders.
- Visible Arterial & Venous Blood columns in Ocular Fundus enables us to observe diverse ways of ocular manifestations of different Hematological Disorders.

Anemias

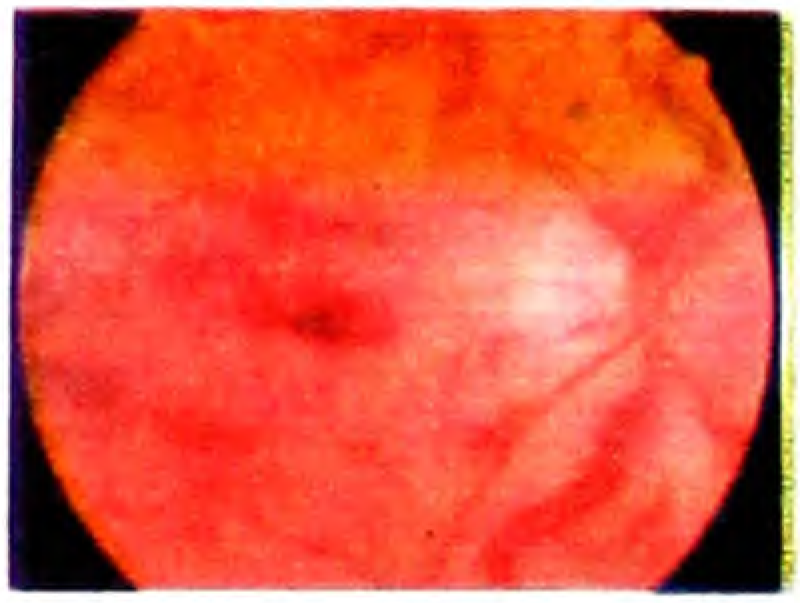
- Generalized pallor of conjunctiva, fundal back ground and optic disc.
- Retinal oedema.
- Dilated retinal vessels.
- Retinal hemorrhages.
- Retinal exudates.
- Retinal detachment.
- Optic neuritis.
- Papillitis.
- Extraocular palsies.
- Extraocular Hemorrhages.
- Lens opacities.

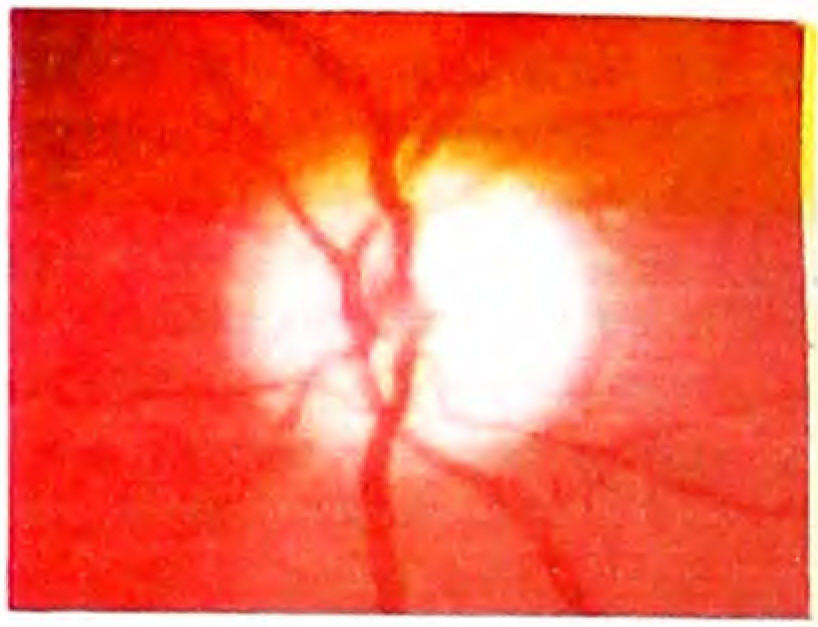




Myeloproliferative Disorders-Ploycythemia Rubra Vera.

- Dilated retinal vessels.
- Retinal hemorrhages.
- Central retinal Venous occlusion.
- Hyperemia of Optic disc.
- Papilledema.
- Optic Nerve atrophy.
- Visual Field Defects.
- Vascular Congestion of Conjunctiva.





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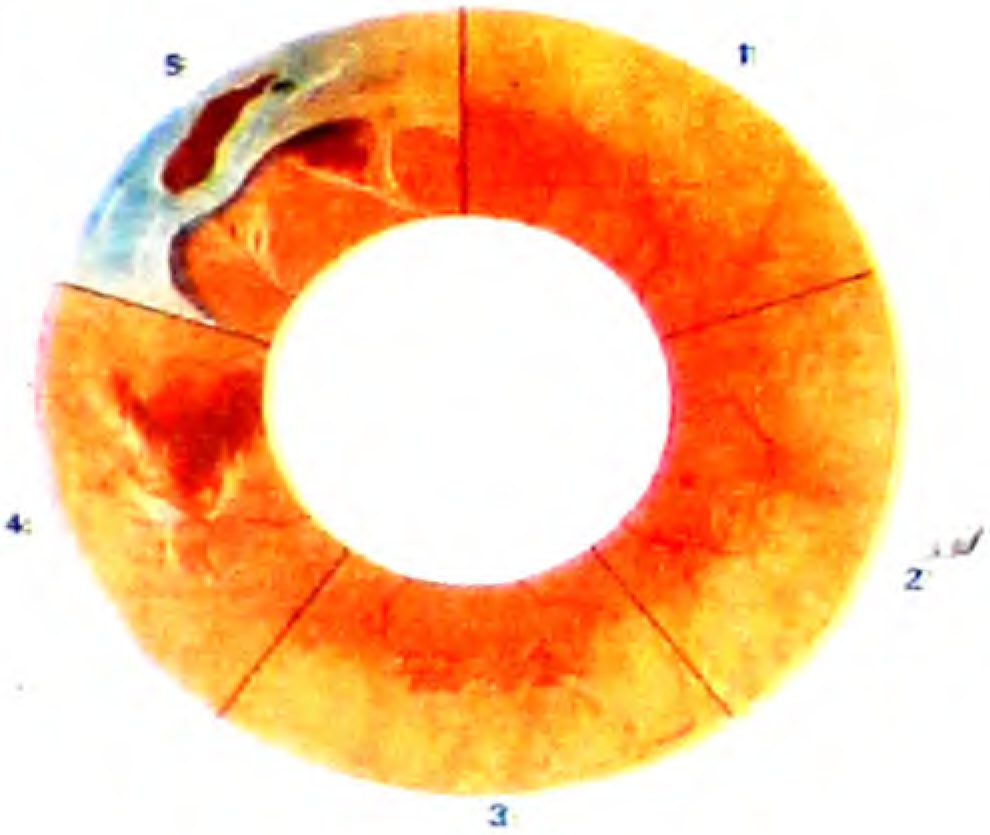
Sickle Cell Disease

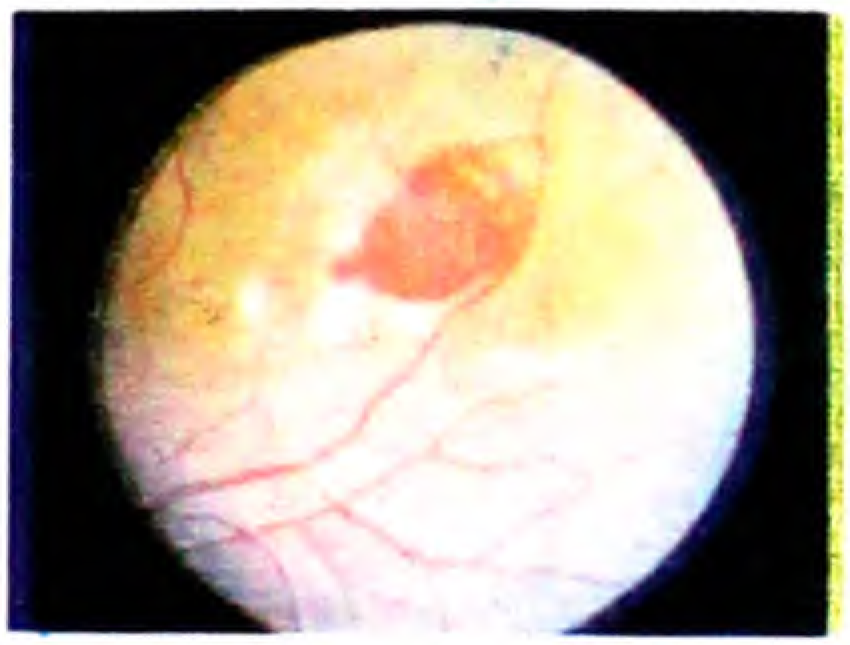
Proliferative Sickle Cell Retinopathy

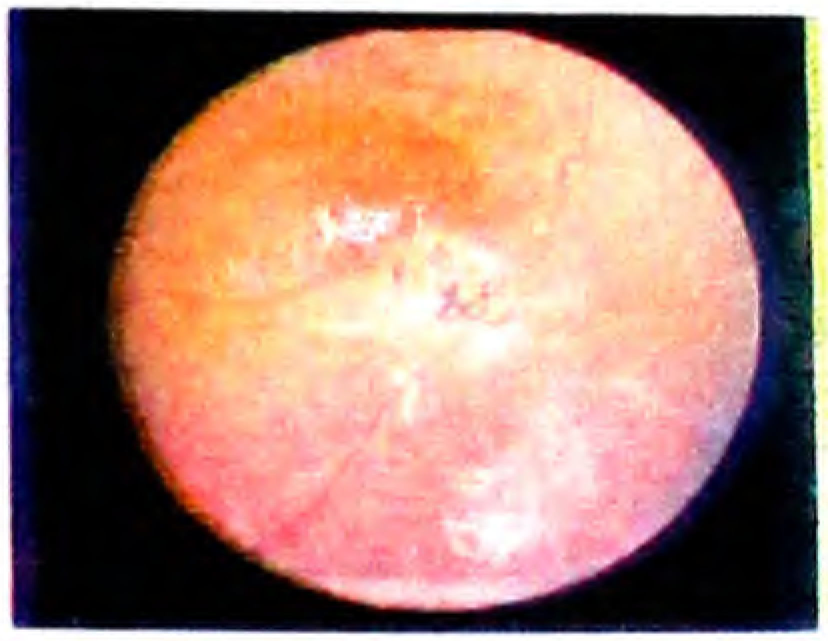
- Stage of peripheral arteriolar occlusion.
- Stage of peripheral arteriovenous anastomosis.
- Stage of neovascularisation (Sea fan).
- Stage of vitreous hemorrhage.
- Stage of vitreoretinal traction bands and tractional retinal detachment.



Non Proliferative Sickle Cell Retinopathy

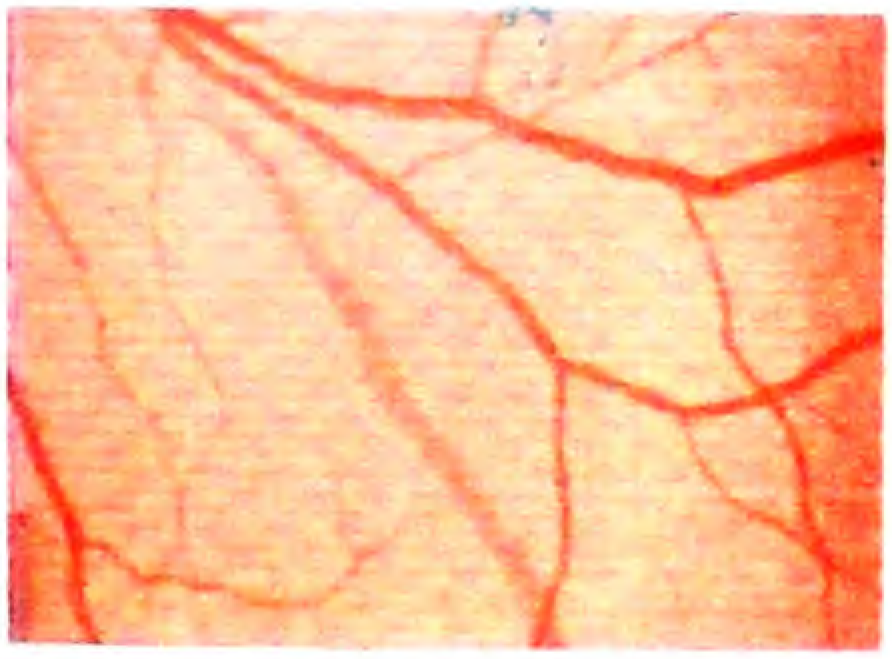
- Venous tortuosity.
- 'Silver wiring' of arterioles in the peripheral retina.
- 'Salmon patches'.
- 'Black sun bursts'.
- 'Macular depression sign'.
- Areas of peripheral whitening of retina.
- Peripheral retinal tears .
- Vitreous hemorrhages or retinal detachment.

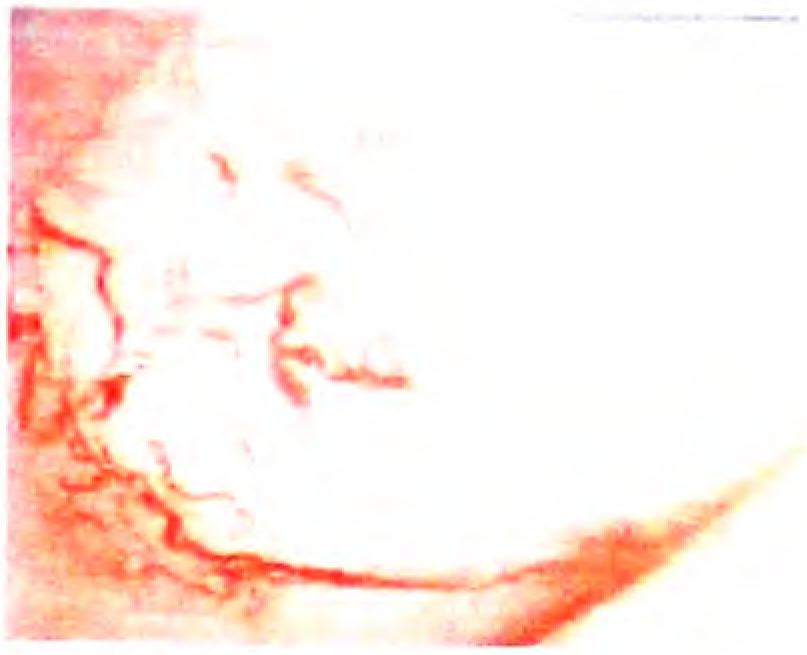




Other Features

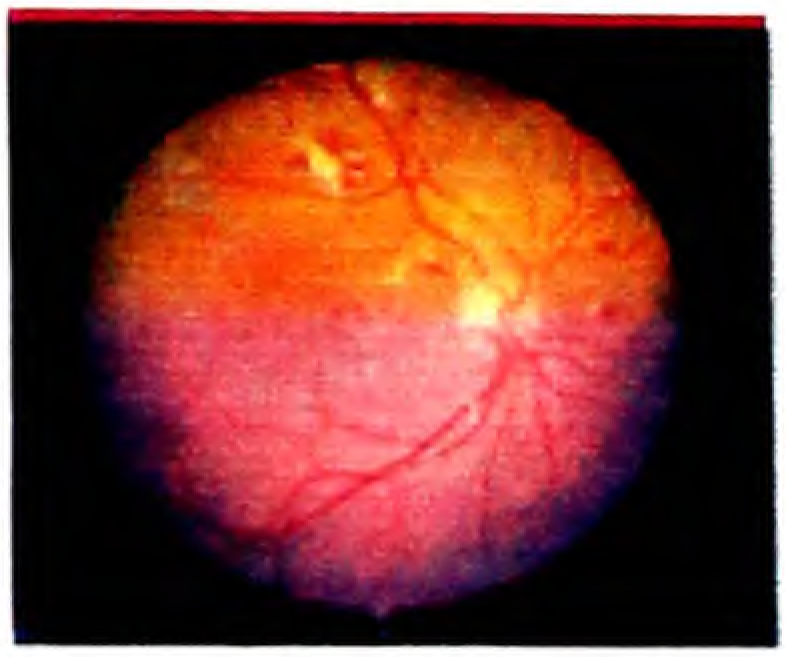
- Macular arteriolar occlusion.
- Central retinal arterial & Veneous occlusion.
- Choroidal vascular occlusion.
- Angioid streaks.
- Comma shaped or corkscrews vessels in conjunctiva.
- Iris atrophy.
- Mild anterior chamber cells and flare.
- Epicanthal folds.

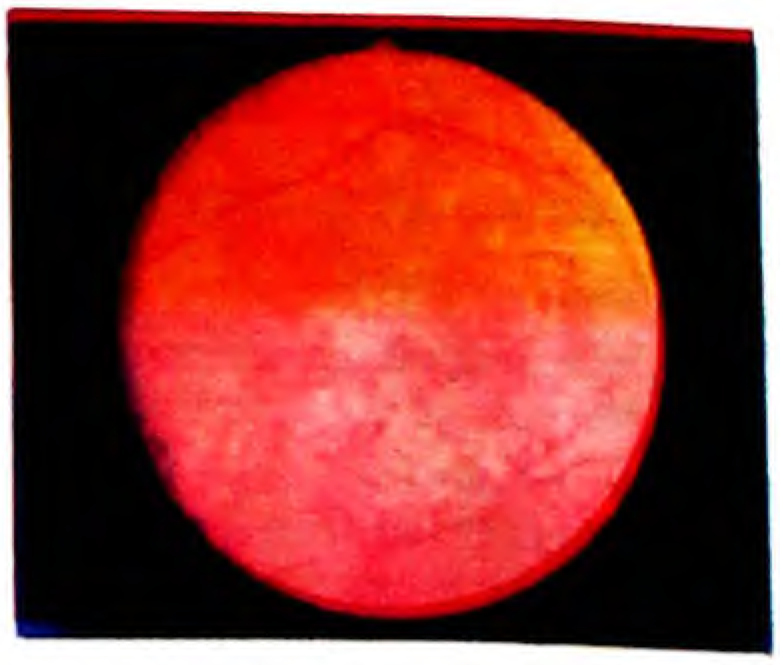




Leukemias

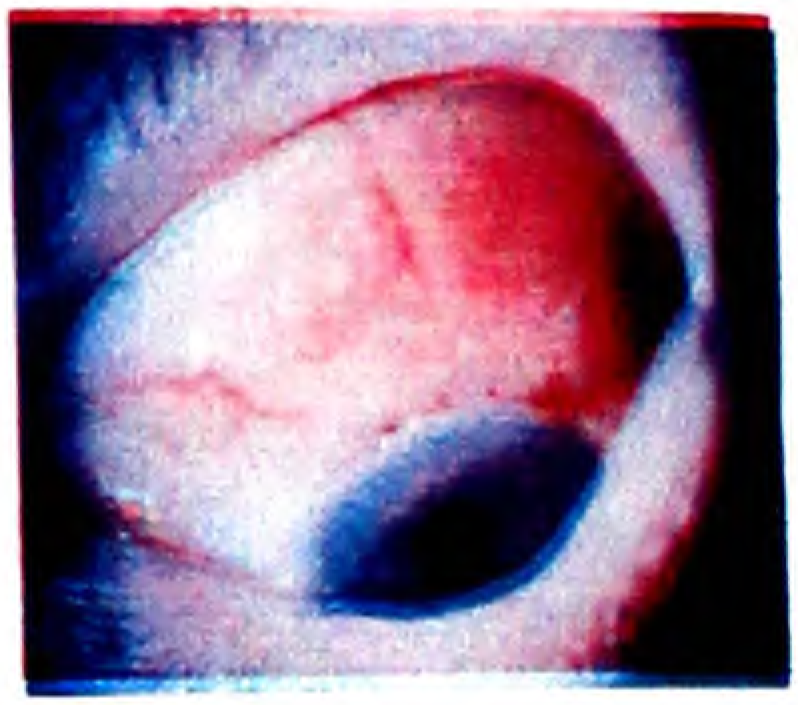
- Retinal hemorrhages.
- Roth spots and cotton wool spots.
- Peripheral retinal neovascularization
- Leukemic pigment epitheliopathy characterized by a 'Leopard Spot Retina'.
- Proptosis.
- Subconjunctival haemorrhages.
- Iris thickening, Iritis and hypopyon.
- Optic neuropathy.
- Cranial nerve palsies.





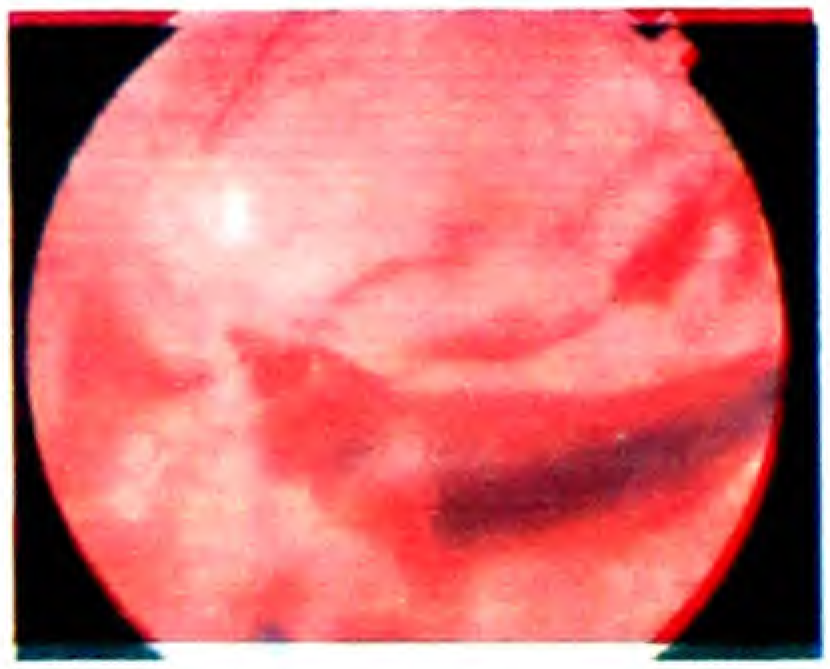
Non Hodgkin and Hodgkin Lymphoma

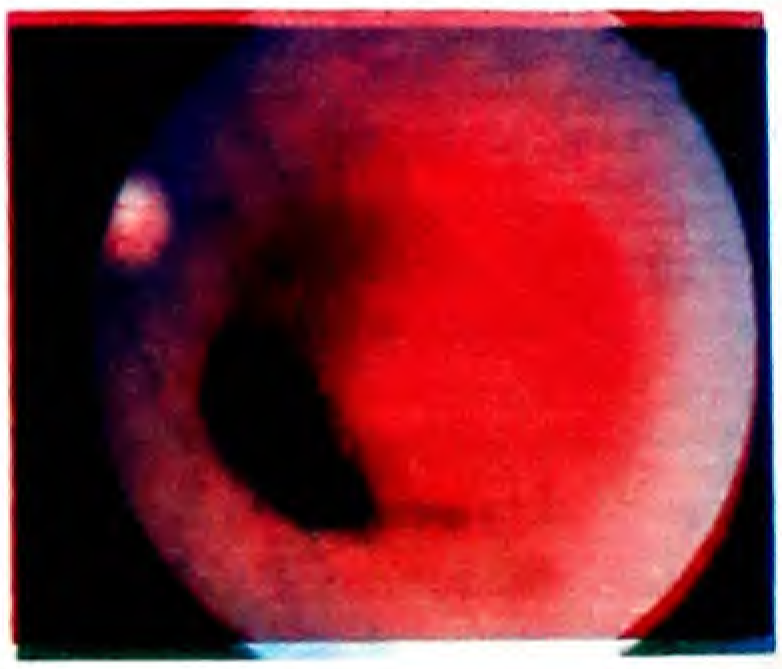
- Painless Swelling of lids.
- Exophthalmos.
- Ocular palsy.
- Uveitis.
- Vitritis.
- Preriphlebitis.
- Chorioretinitis.
- Exudative retinal detachment.
- Optic nerve oedema.
- Extranodal lymphomas of eye.



Thrombocytopenias and Thrombopatheies

- Orbital hemorrhage.
- Subconjunctival hemorrhage.
- Retinal hemorrhage.
- Vitreous hemorrhage.
- Exudative retinal detachment.
- Papilledema.
- Ocular palsies.
- Visual field defects.
- Retinal arterial occlusion.





FNAC OF EWING'S SARCOMA FAMILY OF TUMORS, SKELETAL AND EXTRA-SKELETAL. A STUDY OF 21 CASES.(PART IV)

A.R.Khan

Abstract: *A review of FNAC material over a period of more than 12 years (Jan 91-Feb.2003) revealed 21 cases of Ewing's Sarcoma family of tumors from bone and soft tissues. These included 13 cases from the skeletal and 8 cases from the Extra-Skeletal sites. Those from the skeletal sites included 10 males and 3 females with age range from 4 1/2 to 25 years,, 10 originating in flat bones and 3 in long bones. Smears revealed typical Ewing's Sarcoma in 12 cases and atypical Ewing's Sarcoma in one case of recurrent tumor. The typical Ewing's Sarcoma cases showed 2-cell population of large pale and small dark cells dispersed and in loose clusters. The large cells had pale vesicular nuclei, scanty ill-defined vaculated cytoplasm. The small dark cells intimately mixed with large cells had a small pyknotic nuclei and scanty cytoplasm. The atypical Ewing's Sarcoma showed small, medium and large cells with occasional prominent nucleoli, more cellular cohesion, molding and clustering. In the Extr- Skeletal group there were 4 males and 4 females with age range of 6-25 years, 4 originating from chest wall (including 1 from male breast) 3 from thigh and 1 from deep pelvic soft tissues. Smears from 5 cases (4 from chest wall 1 from pelvic soft tissue) showed a smear pattern labeled as pPNET, characterised by tumor cells, round to elongated and deeply stained nuclei and scanty ill-defined cytoplasm. There was more cell cohesion,, cell molding, clustering, poorly formed and well formed pseudo-rosettes of Wright-Homer type and clean background. The smears in remaining 2 cases from thigh showed a combined pattern of dispersed round vaculated cells of typical Ewing's Sarcoma bone and rosettes of Homer-Wright type accounting for combined smear pattern (EES-pPNET). One case from thigh revealed only blood from a haematoma. The histopathology for comparison was available in 18 cases, 11 from the skeletal tumors & 7 from Extra-Skeletal tumors. In the Extra-Skeletal group 1 case yielded only blood from haematoma superimposed on the tumor. Histopathology confirmed the FNAC diagnosis in all the 18 cases. One case from another state was lost to follow up. The remaining 20 cases were treated. The study proves that FNAC is simple, quick and economic procedure in the diagnosis of Ewing's family of tumors. The diagnosis can be made confidently in the skeletal group of tumors. In the Extra-Skeletal those on the chest wall and the soft tissues the diagnosis can me made confidently. However those in deep soft tissues of abdomen and pelvis require formal biopsy/ ancillary studies for confirmation.*

Keywords: *FNAC : Fine Needle Aspiration Cytology*
E.S : Ewing's Sarcoma Bone. E.E.S : Extra-Skeletal Ewing's sarcoma.
pPNET : primitive peripheral neuro-ectodermal tumor.

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Introduction:

Ewing's Sarcoma of bone (ES), Extra-Skeletal Ewing's Sarcoma (EES) and primitive peripheral neuro-ectodermal tumor (pPNET) are now considered to be the same tumor with variable differentiation with no significant biologic or therapeutic distinction[1-4]. Tumors included in this group are peripheral neuro-epithelioma peripheral neruo-blastoma, extra-osseous Ewing's Sarcoma peripheral neuro-ectodermal sarcoma, para-vertebral round cell tumor in children (Teft's tumor) & malignant small cell tumor of Thoraco-pulmonary origin in children (Askin's tumor)[7-11]. The pPNET refers to the group of neoplasms exhibiting ultra- structural and immuno-histochemical features of neural differentiation and representing primitive neoplasm of neural crest derivation[12]. These tumors are seen in the age group of 11-22 years but have been reported in the newborn and elderly as well. Primary sites include chest wall, trunk, retro-peritoneum, pelvis and rarely extremities[9].

The FNAC of ES, E.E.S and pPNET have been described in the literature [4,13-30]. With a change in the treatment modality of Ewing's- pPNET family of tumors by pre-operative chemotherapy, FNAC can play a useful role as a pre-operative diagnosis with obvious advantages over the open/needle core biopsy.

The author reviews the FNAC experience of 21 such tumors from the skeletal and extra-skeletal sites.

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MATERIAL AND METHODS:

A retrospective and prospective analysis of FNAC over last more than 12 years (Jan, 1991 - Feb, 2003) revealed 21 cases of Skeletal Ewing's Sarcoma, Extra-skeletal Ewing's Sarcoma and primitive peripheral neuro-ectodermal tumors. The aspirations were conducted, with the help of radiographs using 20ml disposable syringes with and without the syringe holder and 23-18 gauge disposable needles. 5-8 slides were preparely, 2-4 slides were stained regularly with MGG and the remaining slides were reserved for special stains/studies.

RESULTS: -

The clinical data of 21 cases of E.S, EES, and pPNET family of tumors is shown in the tables 1A and 1B. The patients presented with pain and/or swelling at the tumor site. In the skeletal group 3 patients presented as cases of osteomyelitis with constitutional symptoms. The age range in the skeletal group was 4 1/2 to 25 years with 10 males and 3 females. In 10 of these cases tumor originated in flat bones and in 3 cases in long limb bones (Table 1A). In extra-skeletal group there were 4 males and 4 females, with age range of 6 to 25 years. Sites of the tumor included 4 from chest wall (including a case from left male breast) 3 from thigh and 1 from the pelvic soft tissues. FNAC diagnosis was made from primary tumors in 11 cases and from recurrent/metastatic tumors in 2 cases in the skeletal group diagnosed earlier by histopatholgy. Smear diagnosis was Ewing's Sarcoma typical in 12 cases and Ewing's Sarcoma atypical in 1 case of recurrent tumor (case 10). FNAC diagnosis was made from primary tumors in 7 out of 8 cases in extra-skeletal group and labeled as pPNET in 5 cases, and EES-pPNET in 2 cases. One case (case 20) yielded only blood due to hematoma superimposed on the tumor. Histopathology for comparison was available in 11 out of 13 cases of skeletal group, which confirmed the FNAC diagnosis. The remaining 2 cases were treated on FNAC diagnosis without formal biopsy. In the extra-skeletal group histopathology was available in 7 out of 8 cases. Remaining case, a 23-year male from Darjeeling left Srinagar for his home for further management and was not available for follow-up. Histopathology confirm the diagnosis in all the seven cases.

Table IA: FNAC Skeletal Ewing's sarcoma (ES/EES/pPNET) family of tumors skeletal group

S.No.	Year	Age/Sex	Site	FNAC
01.	1991	22/M	Iliac bone	E. S.
	1992	Tumor Recurrence	Abdominal wall	E. S.
02.	1991	10/M	Scapula	E. S.
03.	1992	18/M	Iliac bone	E. S.
04.	1994	23/M	Metatarsal	E. S.
05.	1995	15/M	Iliac bone	E. S.
06.	1996	20/M	Iliac bone	E. S.
07.	1996	15/M	Iliac bone	E. S.
08.	1998.	17/M	Meta-tarsal	E. S.
		6 weeks later	Scalp	E. S.
09.	1999	11/M	Tibia	Primary diag.(Histopath)
	2000	Tumor recurrence	Scalp	E. S.
10.	1999	16/M	Chest wall (Ribs)	E. S. Typical
	2002	Tumor recurrence	Ribs & soft tissue	Primary diag.(Histopath)
11.	2000	4 ½/F	Scalp	E. S.
12.	2002	9/F	Humorous	E. S.
13.	2003	25/F	Humorous	E. S.

Table IB: Extra-skeletal group

S.No.	Year	Age/Sex	Anatsite	FNAC
14.	1996	6/F	Thigh	EES-pPNET
15.	1997	17/F	Chest wall	pPNET
16.	1998	15/F	Iliac fossa	pPNET
17.	2000	23/M	Breast	pPNET
18.	2002	15./F	Chest wall	pPNET
19.	2002	9/F	Chest wall	pPNET
20.	2002	16./M	Thigh	Hematoma
21.	2003	25/M	Thigh	ESS-pPNET

Abbreviation.

1. E. S: Ewing's sarcoma typical
2. E.S: Atypical Ewing's sarcoma-atypical.
3. EES- pPNET: Extra-skeletal Ewing's sarcoma.
4. pPNET: Primitive peripheral neuro-ectodermal tumor.

Cytological findings: -

Skeletal groups showed 2 smear patterns labeled as Ewing's Sarcoma-typical (12 cases) and Ewing's Sarcoma - atypical (1 case). Typical Ewing's smear pattern showed cellular smears of round cells of 2 types, the larger and the smaller, dispersed and in loose clusters. The large cells showed fine chormatin, scanty vaculated PAS positive cytoplasm with lace-like pattern in the background at places. The smaller cells had small dense nuclei and scanty cytoplasm (Fig 1-3). The atypical Ewing's Sarcoma smears showed considerable variation in tumor cell size from small, intermediate to large one with prominent nucleoli. The tumor cells were more cohesive with molding and clustering (Fig-4). In the extra-skeletal group, 2 types of smears

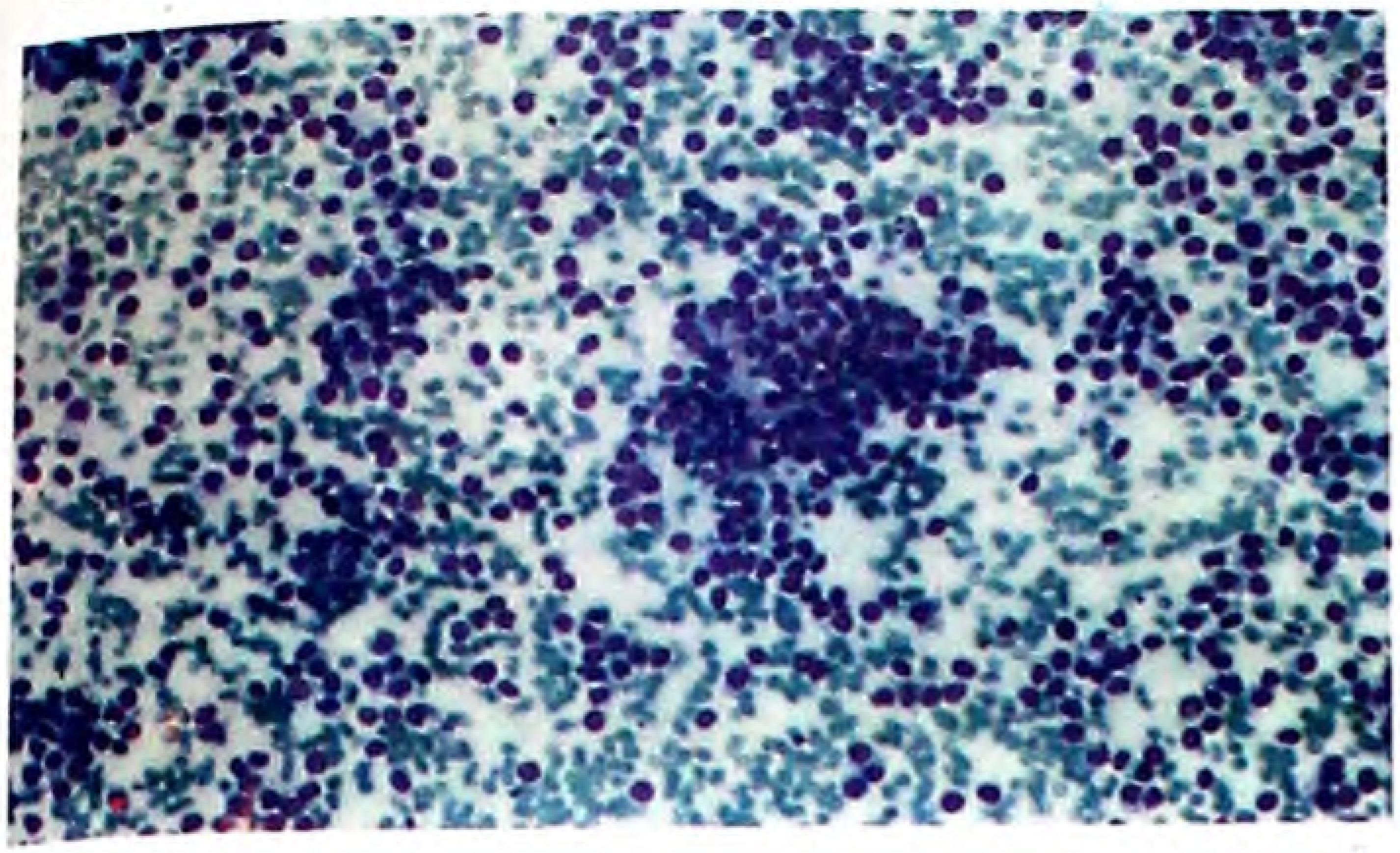

Fig.1. Ewing's Sarcoma Skeletal. Shows loose clusters & dispersed round tumor cells MGG x 200. -

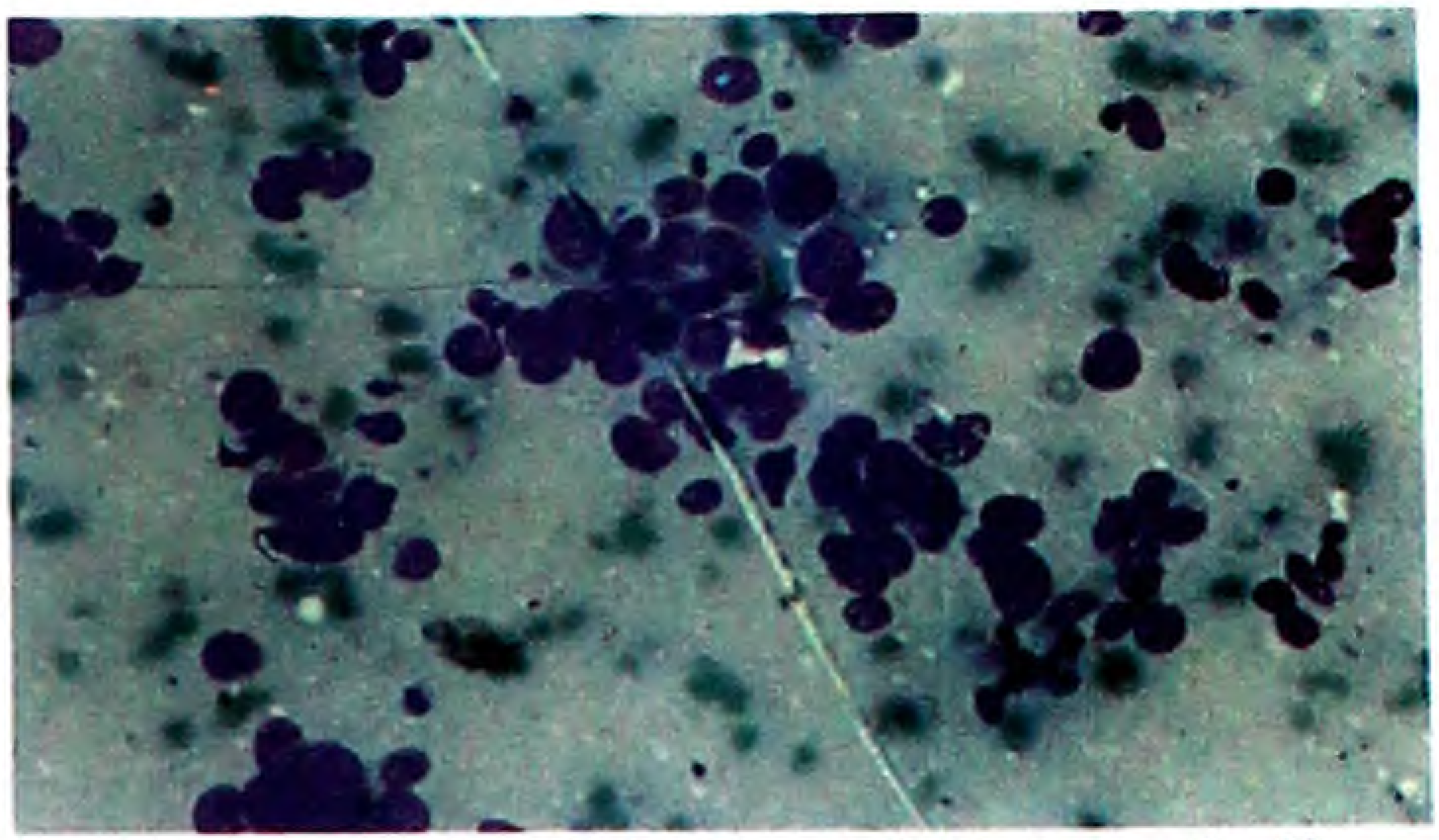

Fig 4. Ewing's Sarcoma Skeletal (Atypical): Shows considerable variation in tumor cells in clusters with molding MGG x 400.

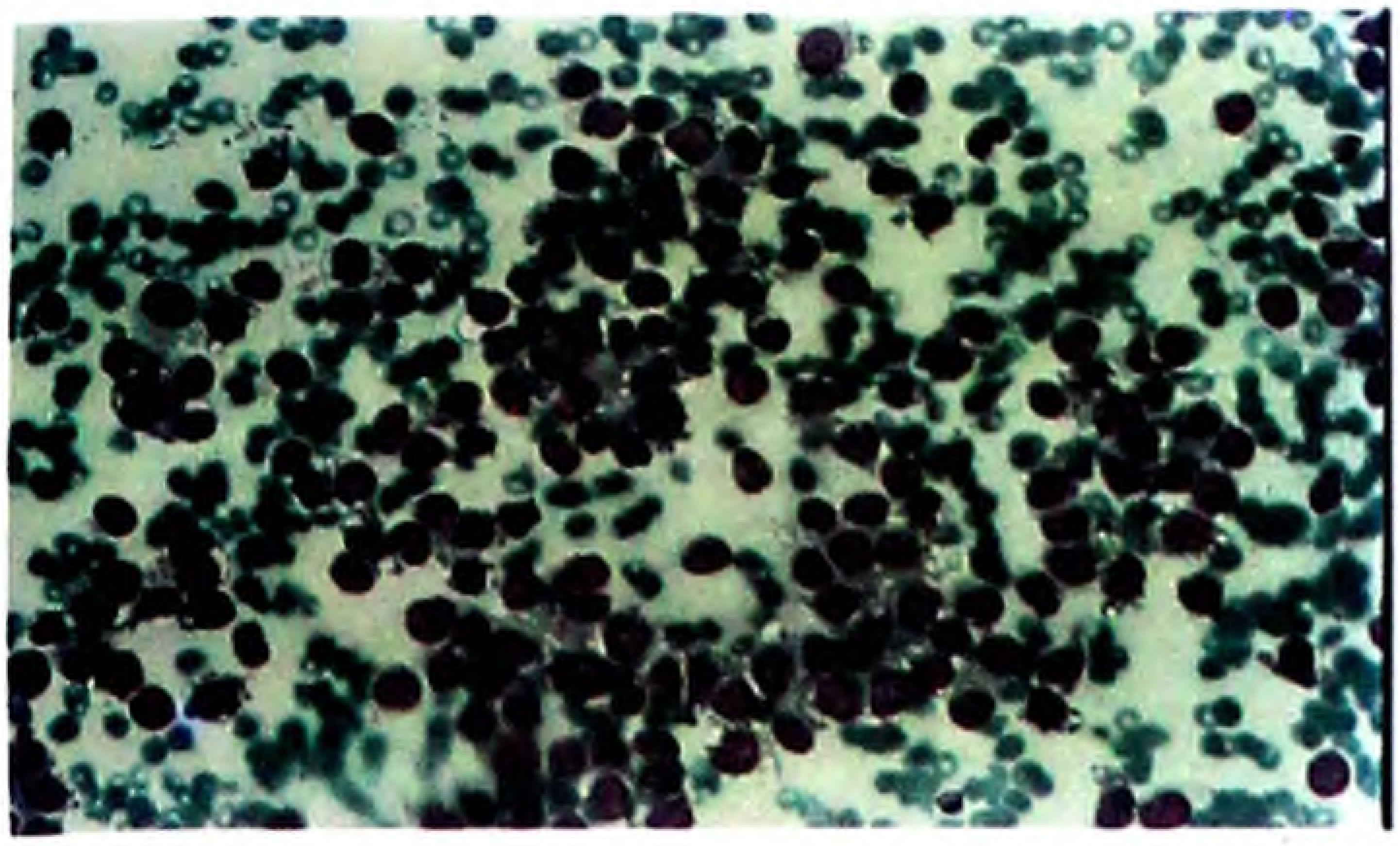

Fig 2. Ewing' Sarcoma Skeletal. Shows dispersed monomorphic tumor cells with vacuolated cytoplasm MGG x 400.

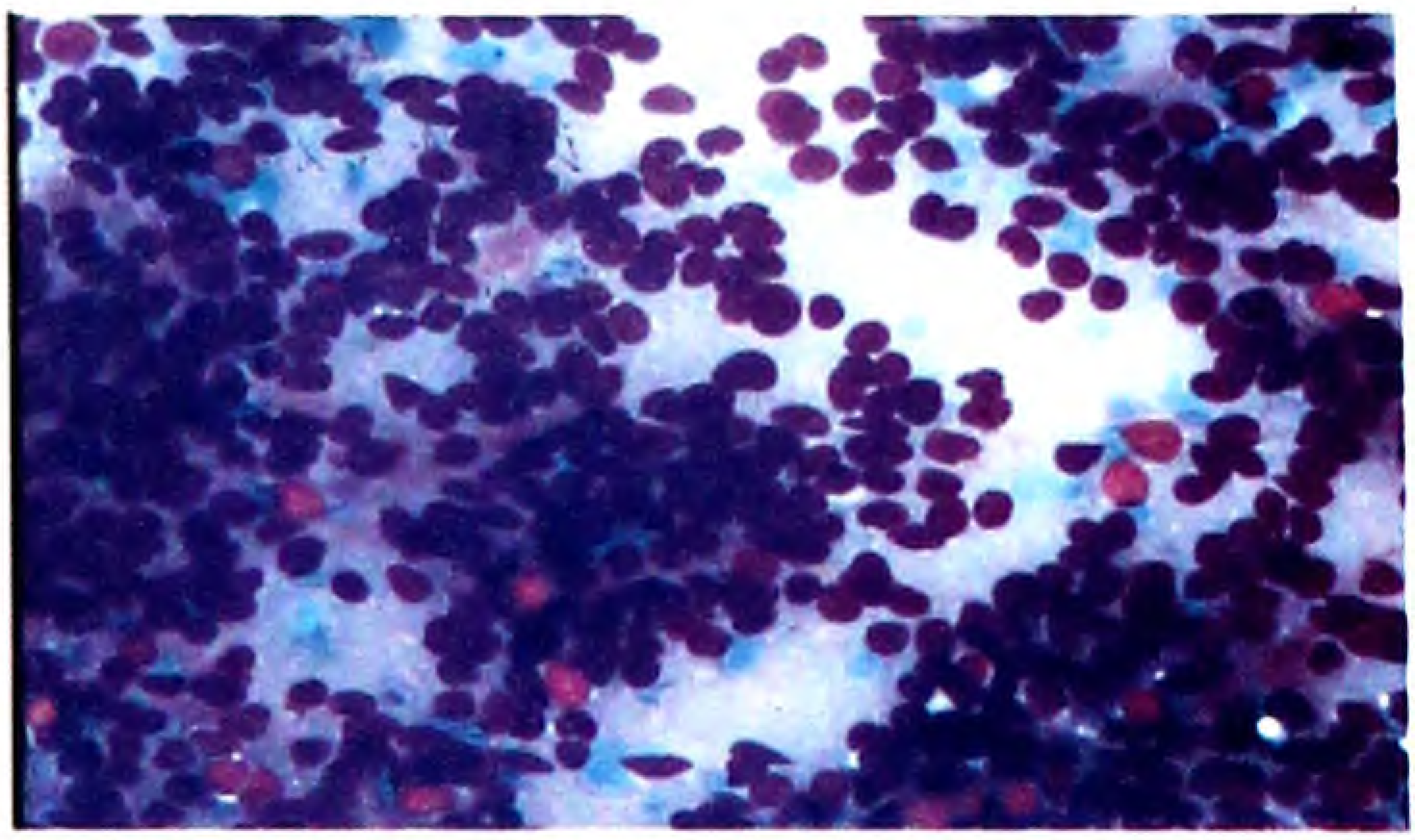

Fig 5. Ewing's Sarcoma Extra- skeletal pPNET smear pattern. Shows clusters of monomorphic round cells MGG x 400.

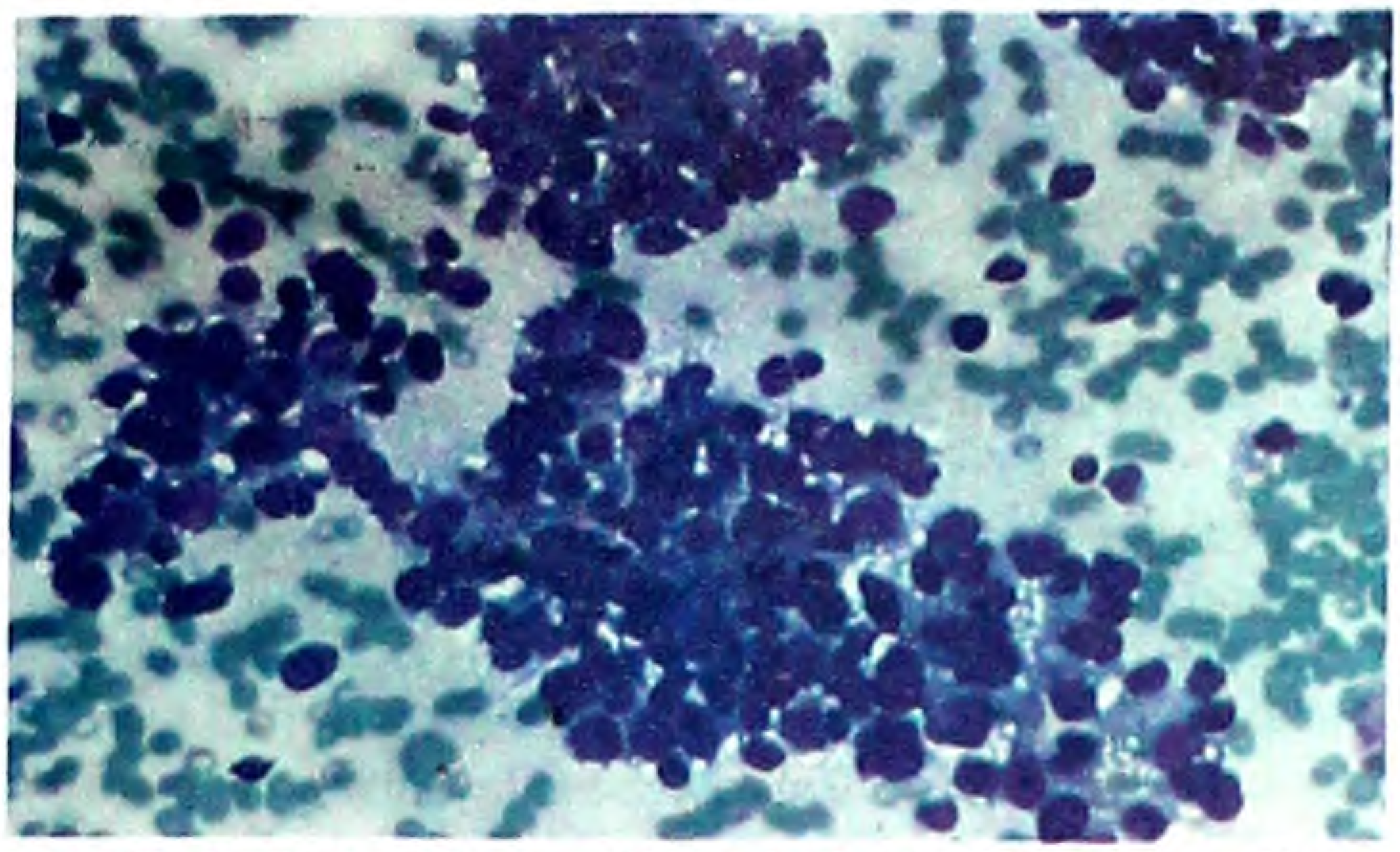

Fig 3. Ewing's Sarcoma Extra –Skeletal pPNET pattern. Shows clusters of tumors cells with ill formed rosettes MGG x 400.

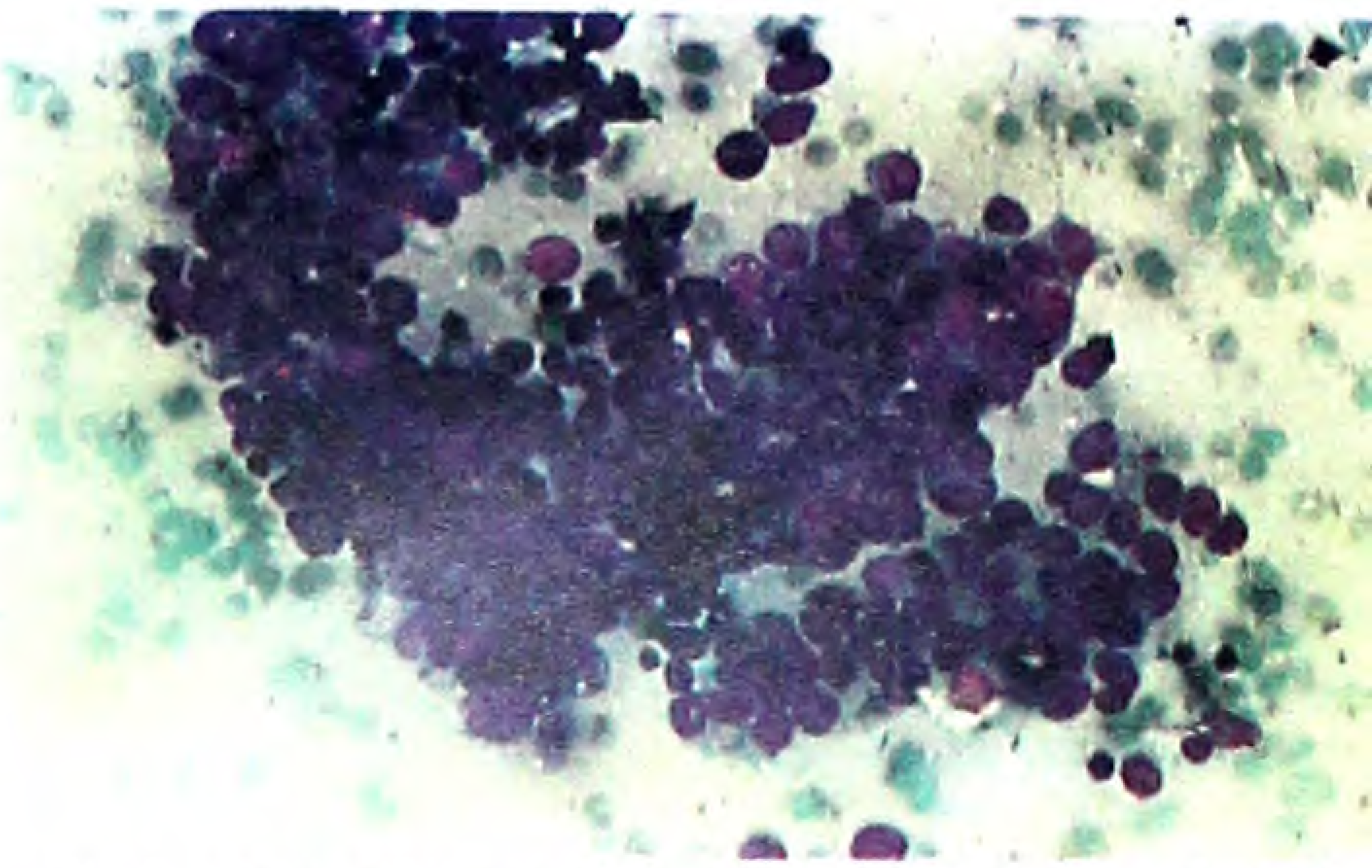

Fig 6. Ewing's Sarcoma Skeletal. Shows clusters of two type of tumors cells large pale & small dark intimately mixed MGG x 400.

patterns observed were labeled as pPNET and EES-pPNET pattern .The pPNET pattern seen in 5 cases (3from chest wall, 1 from male breast and 1 from pelvic soft tissues) showed 2 types of cells, the round to elongated with more deeply stained nuclei, ill defined scanty cytoplasm. Tumor cells however, were more cohesive with striking clustering, molding and pseudorosettes with fibrillary material in the centre. The background was clean (Figs 5-7). The EES-pPNET pattern observed in 2 cases from thigh showed an intermediate picture with dispersed vacuolated cells of skeletal Ewing's Sarcoma and many pseudo-rosettes of Homer-Wright as type observed in pPNET pattern (Fig 8).

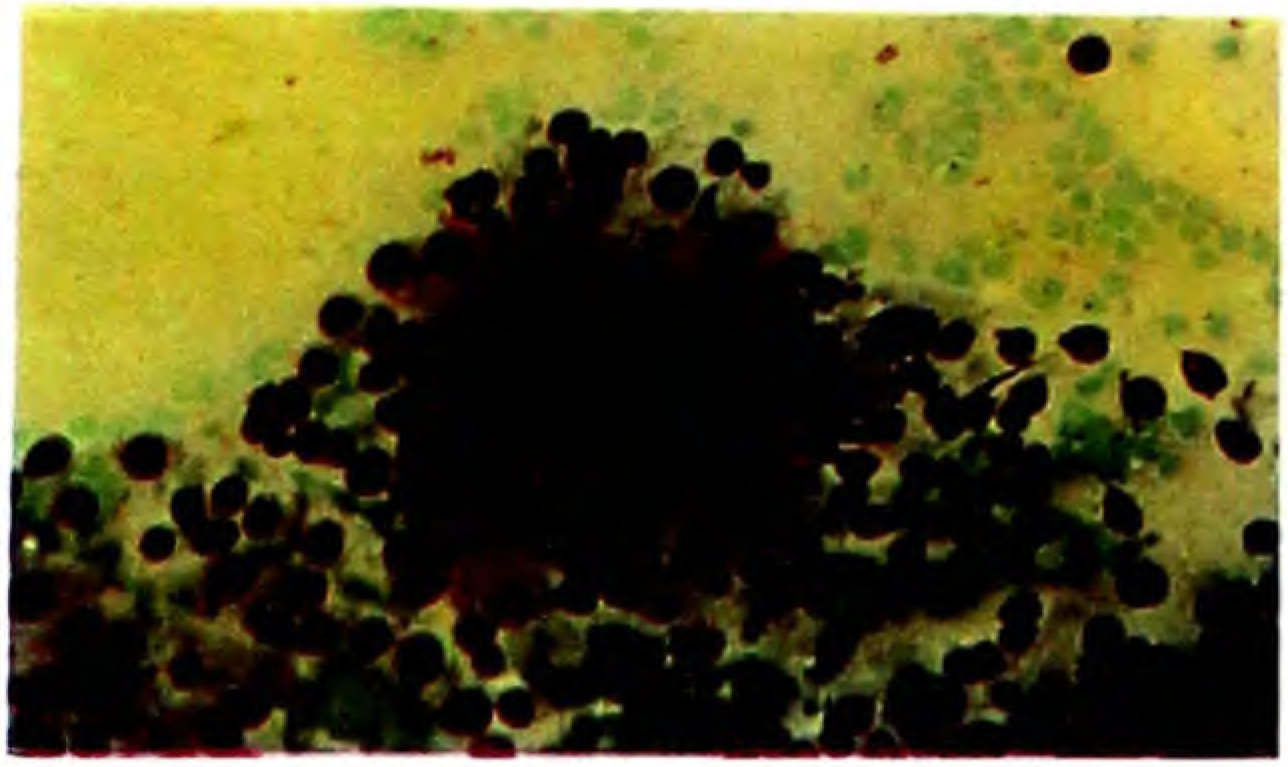

Fig 7. Psudo- rosette from chest wall tumor. Shows purplish fibrillar material in center surrounded by tumor cell MGG x 400.

Discussion:

Ewing's Sarcoma (E.S), extra-skeletal Ewing's Sarcoma (EES) and primitive peripheral neuro-ectodermal tumors (pPNET) are members of a family of tumors of neuro-ectodermal origin[1] Ewing's Sarcoma represents the most undifferentiated & the pPNET, the most differentiated end of the spectrum.[2-4] The two ends of the spectrum differ in morphology as well as in neuronal differentiation.[5] However in addition to common hitogensis, this family of tumors share the common cytogenetic & molecular changes[21-34].which involve the translocation of Ewing's Sarcoma gene on chrosome 22(22q12) on to a number of other genes like Fli-1 on chromosome 11(11q24) in 90% of cases & ERG on chromosome21(21q22) ,ETVI on chromosome 7 (7p22) & rarely others. The tumors also share expression of glyycoprotein surface antigen p30/32 (mic[2])/CD99, a cell membrane protein of unknown function that is the product of a pseudo-autosomal gene located on the short arm of the sex chromosomes[35].This translocation is shared by olfactory neuro-blastoma & mesenchymal chondro-sarcoma[36] ,which can easily be differentiated otherwise from the Ewing's sarcoma family of tumors. However CD99 expression is not specific for Ewing's sarcoma family of tumors but is shared by a number of tumors both in children & adults[36-40] .CD99 expression is recognized by various monoclonal antibodies 013,HBA71& 12E7 & sensitivity of 98% is reported[40-44] The evaluation of the fusion products of the specific chromosomal translocations t(11;22) (q24;12) & t (21;22) (q22;q12) by RT.PCR is more specific .However, most of these tumors can be diagnosed on morphology alone particularly plastic sections in the context of clinical, laboratory & X Ray details[20,41] and only a few cases require immuno-histochemistry & ultra –structural studies[45] . Morphology in FNAC smears is much better appreciated than in paraffin-embedded sections, without resorting to plastic embedded section to save the time & cost of the procedure. The FNAC of ES-EES typical & atypical variants & pPNET has been reported in the literature.[4,13-30,46]. The author reviewed 21 such tumors over a period >12 years. The smears from skeletal sites irrespective of typical and atypical Ewing's sarcoma with the past history of tumor diagnosis by histopathology in the atypical Ewing's sarcoma were fairly characteristic to offer the diagnosis. In the Extra-Skeletal group the smears from the chest wall, male breast and thigh were again characteristic to offer the correct diagnosis. In the tumor from the pelvis tissues smears were suggestive and demanded confirmation by open biopsy/ancillary studies.

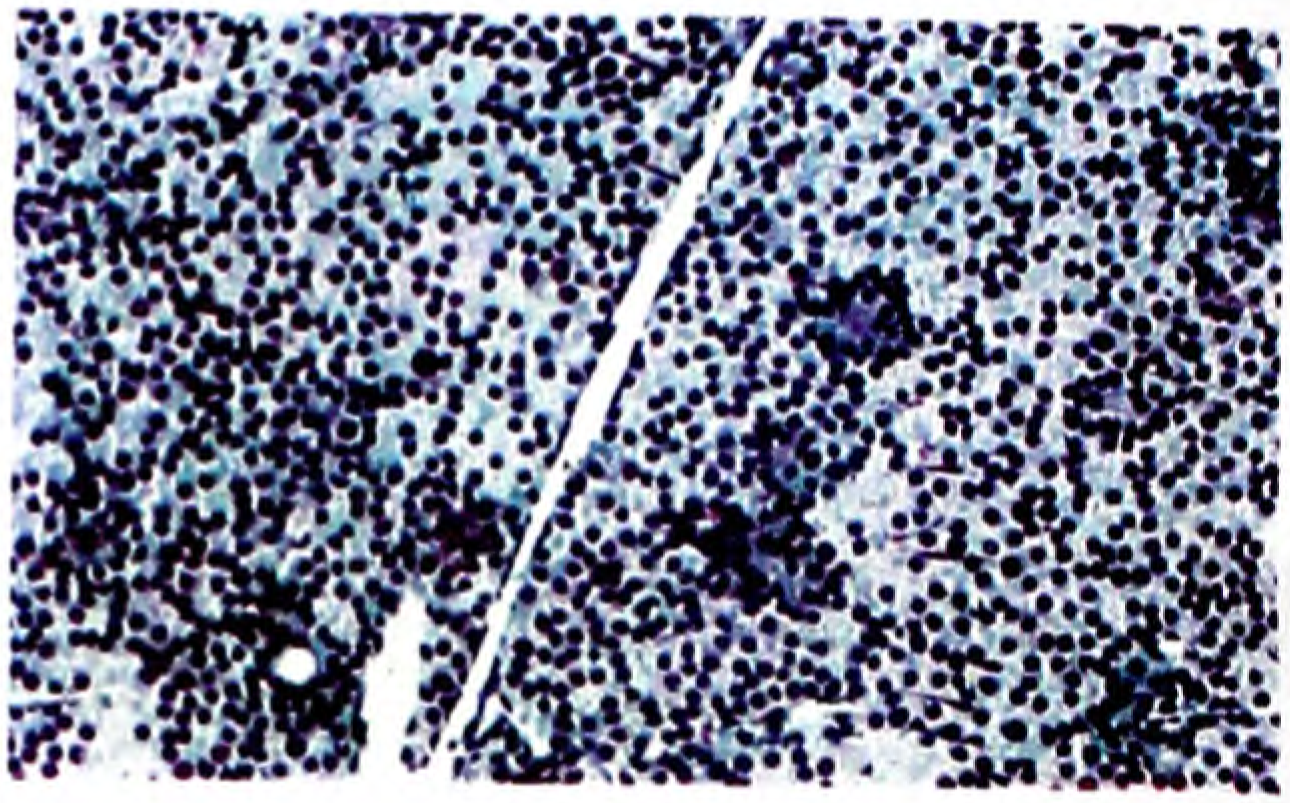

Fig 8. Ewing's Sarcoma Extra-Skeletal from thigh. Shows dispersed monomorphic round tumor cells like typical Ewing's Sarcoma Skeletal & many pseudo- rosettes like Extra-Skeletal Ewing's Sarcoma (EES –pPNET Pattern) MGG x 200.

The differential diagnosis on FNAC as on histopathology includes other round cell tumors like metastatic neuroblastoma, rhabdomysarcoma and NHL in children & metastatic small cell ana-plastic carcinoma & NHL in adults. Smears of neuroblastoma show smaller cells with high N/C ratio, Homer-Wright rosettes & neurophil matrix. The undifferentiated form of this tumor however cannot be distinguished from pPNET on morphology alone without ancillary studies. Rhabdomyo sarcoma smears show clusters & dispersed tumor cells more pleomorphic with small & large cells including some spindle & multinucleate ones with scanty to abundant dense cytoplasm & more coarer chromation & more prominent nucleoli. Some cases may show myxoid or bubbly appearance in the background due to glycogen rich cytoplasmic fragments. Smears of the non- Hodgkin's lymphoma may show monomorphic round cells with scanty basophilic cytoplasm in lymphoblastic lymphoma or mixed large & small cells in large cell lymphoma with dispersed cells & some cell clusters may be seen. However numerous lympho-glandular bodies are clue to the diagnosis. The metastatic small cell anaplastic carcinoma show small to medium sized tumor cells with scanty, cytoplasm coarser chromative, indistinct nucleoli, nuclear molding, cell clusters,, nuclear breakdown & mitotic figures. The PAS positively reported in 2/3rd of typical Ewing's sarcoma is also reported in 10-50% of pPNET and rhabdomysarcoma and therefore, hardly of any help in differential diagnosis.

The division into skeletal and extra-skeletal tumors appears more relevant than sub typing into Ewing's, EES-pPNET and pPNET. The skeletal Ewing's tumors are usually typical, Atypical variant constituting 5% of tumors[47]. The pPNET of the bone is rare. In the Soft tissues the tumors are invariably of pPNET morphology. The Skeletal Ewing's sarcoma are known to have a 5 year survival of 75% [35]. The Skeletal Ewing's atypical tumor is believed to have some outcome as the typical tumors[44] . The Extra-Skeletal pPNET on the other hand have 20-30% survival[48]. This outcome between Skeletal & Extra-Skeletal Ewing's family of tumors, therefore, may not be dependent on morphologic differentiation, but related to large size, poor circumscription & deep location of Extra-Skeletal tumors. The outcome has also been related to type of fusion. The tumors with EWS/FL-I exon 7/6-fusion type (type I) have a better prognosis than those with EWS/FL-I exon 7/5-fusion type (type II)[32].

In summary, therefore, FNAC is very useful, economic & quick procedure in the diagnosis of Ewing's sarcoma family of tumors. In authors experience accurate diagnosis can be made in Skeletal tumors irrespective of differentiation typical or atypical on the morphology alone. In the Extra-Skeletal sites superficial tumors in the Chest Wall (Askin's tumor) or in lower limbs can also been diagnosed with fair amount of confidence. The success in the deep Soft tissue, para-vertebral & pelvis would depend on the yield of the material & would need biopsy/ancillary studies for confirmation in scanty material & atypical cases. FNAC is also useful in long follow up of there cases for diagnosis of recurrence or rarely intercurrent second malignancy[49,50]

References:

1. Ushishigoma S Shimoda T Nikaido T . Primitive neuro-ectodermal tumors of bone & Soft tissues with reference to histologic differentation in primary or metastatic foci. Acta Pathol, jpn 1992; 42:483-93.
2. Gonzalez- Campora R. Otal-Salaverri e Flores PP. Hevia-Vazquez A.Pascual A G Diez .VSM: Fine needleaspiration of peripheral neuroepithelioma of tissue. Acta Cytol 1992, 36:152-158.
3. Grier H E Ewing's family of tumors: Ewing's sarcoma & primitive neuro-ectodermal tumors Paediatric Clin North Am 1997; 44: 991- 1004.
4. Kilpatrick SE Geisinger KR. Soft tissue sarcomas the usefulness & limitations of fine – needle aspiration biopsy. Am J Clin Pathol 1998; 110: 50-68.
5. Siebonrock KA Nascimento AG Rock NG Comparison of soft tissue. Ewing's sarcoma & peripheral neuro-ectodermal tumor.Clin Orthop Rel Res 996; 329: 288-89.
6. Terrier P Henry- Amar M Triche TJ Horowitz ME Terrier Lacombe MJ Miser JS etal. Is neuroectodermal differentiation of Ewing's sarcoma of bone associated with an unfavourable prognosis? Eur J Cancer 1995;31A:307-14
7. Teft M, Vawter GF Mitus A. Para-vertebral "round cell tumors" in children. Radiology 1969; 92;1501-1509.
8. Angervale L Enzinger FM. Extraskeletal neoplasm resembling Ewing's sarcoma. Cancer 1975;36:240-251
9. Askin FB, Rosai J . Sibley RK . Deher LP : Malignant small cell tumor of the thoracopulmonary region in childhood. Cancer 1979;43 ;2438-2451.
10. Schmudt D. Herman C Jurgens H Harns D. Malignant peripheral neuro ectodermal tumor and its necessary distuction from Evings sarcoma. Cancer 1991 ;68 : 2251-2259.
11. Dehner L P Primiture neuro ectodermal tumor & Ewing's sarcoma Am J Surg Pathol 1993; 17: 1-13.
12. Neuhold N Artlieb U Winimor M Krisek I Schratter M : Aspiration cytology immunocytochemistry and electron microscopy of malignant peripheral neuro-ectodermal tumor. A case report Acta Cytol 1989;33:74-79
13. Reif RM Ewing's sarcoma, cytology of large cell type Acta Cytol 1980;24:175-6
14. Fanning TV Katz RL Ayala AG; Fine needle aspiration of Ewing's sarcoma bone: an analysis of 57 specimens. Acta Cyta! 1985;29:932.
15. Akhter M Ali MA Sabbah R . Aapiration cytology of Eving's sarcoma: Light and electron microscopic correlation . Cancer 1985;56:2051:2060.
16. Dahl I Akerman M Angervale L . Eving's sarcoma of bone : a correlative cytological and histological study of 14 cases. Acta Pathol Microbiol Immunol Scand(A). 1986:94:363-369.
17. Brehaut LE Anderson LH Taylor LH. Extraskeletal Ewing's sarcoma :diagnosis of a case by needle aspiration cytology Acta Cytol 1986;30:683-87.
18. Layfield LJ Glasgow BT Anders KH Mirra JM , Fine needle aspiration cytology of primary bone lesions . Acta Cytol 1987;31:177-184.
19. Anger M Bedard YC Keating S: Diagonis of a peripheral neuro-ectodermal tumor by fine needle aspiration cytology. Cytopathology 1990;1:243:-249.
20. Christie DR Bilous KM carr PJ Diagnostic difficulties in extra-osseas Ewing's sarcoma. A proposal for diagnostic criteria. Australas Radiol 1997;41:22:-28.
21. Mandal A Misra Dk Ewing's sarcoma of bone a study of 71 cases by fine needle aspiration cytology. J Ind Med association 1996;94:135-137.
22. Renshau A-A Perez- Atayde AR Fletcher JA Granter SR

Cytology of typical and atypical Ewing's sarcome /PNET: Am J Clin Pathol 1996;106:620-624.

23. Das DK Bhambhani S Chachra KL Murthy NS Tripathi RP : Small round cell tumors of the abdomen cytologic features in the diagnosis and differential dignosis. ActaCytol 1997;41:1035:1047.
24. Bakhos R Andrey J Bhoopalam N Jensen J Reyes CV. Fine needle aspiration cytology of extra-skeletal Ewing's sarcoma Diagn Cytopathol 1998;18:137:-40.
25. Layfield LT Liuk Dodge RK . Logiste regression analysis of small round cell neoplasm. A cytologic tudy. Dign. Cytopathol 1999;20:271:277.
26. Crist WM Kum LE : common solid tumors of childhood N Eng J Med 1991;624:324
27. Sahu K Pai PP Khadikar UN. Fine needle aspiration cytology of the Ewing's sarcomafamily of tumors Acta Cytol 200;44:332:336
28. Horowitz ME Delaney TF Malawer MM Tsokos NG Ewing's sarcoma of bone and soft tissue of thew peripheral primiter neuroectodermal tumor . In principles and Practice of Pediatric oncology .2nd Eoited by PA Pizzo DG Poplack Philadelphia, JB lippincott 1993;pp295-821.
29. Guiter GE Gambone MY Zakowski MF. The cytology of Extra-skeletal Ewing's sarcoma cancer Cytopathol 1998;86(3):141-148.
30. Jambhekar N Khatib Y. Expectations and limitations. A review part II J of Cytology, 200; 18(2):57-72.
31. Landany M Heinenmann FS Huros AG Rao PH . Chen Q. Jhanwar SC. Neural differentiation in small round cell tumorebone and soft tissue with the translocation t (11;22)(q24;q12) : an immunohistochemical study of 11 cases. Human pathol 1998; 21:1254-3.
32. De Alava E. Kawai A Healey JH , Fligman 1, Meyers P. Huvos AG etol EWS- FLI fusion transcript structure is an independent determinant of prognosis in Ewing's sarcoma. J clin Oncol 1998;16:1248-55.
33. Stephenson CF Bridge JA Sandberg AA Cytogenetic and pathologic aspects of Ewing's sarcoma and neuroectodermal tumors. Human pathol 1992;29:1270-72.
34. Dalattre O Zucman J Melof T etal . The Ewingls family of tumors- a suligroup of small round cell tumors defined by specific chimeric transcripts. N Eng J Med 1994;333:294-99.
35. Rosai J Ackerman's surgical pathology. St louis: Mosby New york 1996:2103.
36. Grater SR Renshan AA Fletcher CD Bhan AK Fosenborg AE.DEaa reactivity in mesenchymal chondro-sarcoma Human Pathol 1996; 27: 1273:-6.
37. Andrassy RT Pediatric Surgical Oncology Philadelphia WB. Saunders 1998:425-6
38. Fellinger EJ Garin-Chesa P Triche TJ Huvos AG Retting WJ Immunohistochemical analysis of Ewing's cell surface auligen p 30/32 (mic2) Am J pathal 1991;139:317-325.
39. Rumani P Rampling D lind M. immunocytochemicel study of CD99 in small round cell tumors of childhood an assessment of its sensitivity and specific Hitopethology 1993;23:557-561.
40. Weidnar N Tgoe J. Immunohistochemical profile of monoclonal antibody OB: antibody tht recognices a glycoprotein p30/ 32MIC2and is useful in diagnosing Ewing's sarcoma and peripheral neuroepithelioms (see comments).Am J Sung pathal 1994;18:486-494.
41. Nascimento A-G Unii KK Pritchord DJ. Cooper KL, Dahlin DC. A clinicopathologic study of 20 cases of large cell (atypical) Ewing's sarcoms of bone. Am J Sung pathol 1980;4:29-36.
42. Stevenson AJ Chatten J Bertoni F. Mieltinen M. CD99 (p 30/ 32MIC2) neuro ectodermal Ewing's sarcoma antigen as on immnohist 1994;2:231:-40
43. Bhan AK Immunohistochemistry of neoplasia Diagnostic stratigies on differentiation anligeus . In Diagnostic Immunopathology 2nd Edition. RB Column , AK Bhan and RT Mc Clusky , Editors 1995, RavenPress New york p 445-478.
44. Collins BT Cramer HM MC Gahey Frain BE Dairs HM. Fine needle aspirations biopsy of metastatic Ewing's sarcoma with MIC2(CD99) immunohistochemistry. Diagn Cytopathal 1999;19(5)382-84.
45. Silverman JF Dabbs DT Ganick DT Holbrook CT Geisinger KR: Fine needle aspiration cytology of neuro blastoma including perpheral neuro ectodermal tumor with immunocytochemical and ultra structural confirmation Acta Cytol 1998; 32:367-376.
46. Kumar PV: Fine needle aspiration finding in malignath small cell tumor of thoraco- pulmonary region (Askin tumor) Acta Cytol 1994;38:702:706.
47. Unni KK Inwards CY : Tumors of osteoarticular system . In Diagnostic Histopathology of Tumors Ed. Chritopher DM fletcher II Edition Churchill Livingstone 2000. pp 1541-1606.
48. Fletcher CDM Peripheral neuroectoderlmal tumors. In Diagnostic Histopathology of tumors Ed. Fletedher CDM II Edition . Churchill livingstion pp 1698-1700.
49. Bacei G Fervari S Barvieri E etal long-term follow up for patients with Ewing's sarcoma of bone treated with adjuvant and neo-adjuvant chemotherapy updated results of 3 sequential studies. Oncol Res. 1997;4:977-985.
50. Kuttesch JF Wexter lh Marcus RB etal. Second malignanoy in Ewing's sarcoma radiation dose dependency of secondary sarcoma. J Clim Oncol1996;14:2818-2825.

RECOMBINANT DNA THERAPY IN MEDICINE

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Introduction:

Recombinant DNA technology has revolutionized biology and is having an ever increasing impact on clinical medicine. Much has been learned about human genetic disease from pedigree analysis and study of affected proteins, but in many cases where specific genetic defect is un- known, these approaches cannot be used. The new technology cir- cumvents these limitations by going directly to the DNA molecule for information. Manipulation of a DNA sequence and the construction of chimeric molecules, so-called genetic engineering, provides a means of studying how a specific segment of DNA works.

History:

1651 - William Harvey proposed the idea that all animals develop from eggs.
1677 - Antoni van Leeuwenhoek believed that sperm contained a child in miniature, which grew larger inside the female's body.
1866 - Gregor Mendel published his groundbreaking studies on inheritance in peas.
1879 - Herman Fol and Oscar Hertwig observed the union of egg and sperm in animals.
1889 - August Weismann believed that variations in offspring result from the union of a substance from the parents.
1944 - Oswald T. Avery demonstrated that DNA carried the ge- netic information in bacteria.
1953 - James Watson and Francis Crick proposed the now-famous double-helix model of DNA.
Mid-1980s- Human genome project was initiated.

Recombinant DNA Technology: Recombinant DNA technology is genetic engineering which effects artificial modification of the genetic constitution of a living cell by introduction of foreign DNA through ex- perimental techniques.

Tools of Recambinant DNA Technology: The various "biological tools" used to bring about genetic manipulations are -

A. Enzymes e.g. Restriction Endo nucleases, Exonucleases etc.

B. Passenger DNA: foreign DNA (insert DNA fragment) which is passively transferred from one cell to another cell of organ is known as passenger DNA. e.g. cDNA (Complimentary DNA), synthetic DNA etc.

C. Vector or vehicle DNA: The DNA which acts as the carrier is known as the vector or vehicle DNA. e.g. Bacterial plasmids, Bac- teriophages, Cosmids etc.

Stages of Recombinant DNA Technology:

1. ***Isolation of specific DNA (insert DNA):*** The genomic DNA is very large in size. Isolation of a specific fragment of DNA can be achieved by splicing or cleaving brought about by a group of key enzymes called "restriction endonucleases".

   Restriction endonucleases: (They are compared to chemical knife). Certain endonucleases, enzymes that cut DNA at specific DNA sequences within the molecule are a key biological tool in recombinant DNA technology (Genetic engineering).

II. Chimeric or Hybrid DNA: The main aim of genetic engineering is to insert DNA of interest (passenger DNA) into
a vector DNA so that the DNA fragment replicates along with the vector after annealing. This hybrid combination of two fragments of DNA is referred to as

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chimeric DNA or hybrid DNA or recombinant DNA.

Steps for preparation:

- A circular plasmid vector is first cut with a specific *"restriction endonu- clease"*. If Eco R I is used, sticky ends with TTAA sequence on one DNA strand and AATT sequence on the other strand are produced.
- The human DNA (passenger or insert DNA) is also cut with the same restriction endonuclease, so that the same sequences are produced on the sticky ends of the cut piece.
- Next, the vector DNA and human cut piece DNA are incubated together so that *"annealing"* occurs. The sticky ends of both vector and human DNA have complementary sequences hence they come into contact with each other.
- The enzyme *"DNA Ligase"* is allowed to act on the hybrid or chimeric DNA molecule. The enzyme joins the two fragments by convalent phosphodiester linkages between the vector and insert molecules.

III. Cloning of Chimeric DNA:

What is a clone ?

A clone is a large population of identical molecules, bacteria or cells that arise from a common ancestor.

This cloning allows for the production of a large number of identical DNA molecules which can then be characterized or used for various purposes.

The chimeric DNA contained in a plasmic vector or phages or cosmids can be introduced into bacterial cells *E.Coli* strain C 101 by a process called *transfection.* The replicating bacterial cell (host cell) permits the amplification of the chimeric DNA of the vector.

In this way, cloning results in the production of large number of identical target DNA molecules. The cloned target DNA is released from its vector by cleavage using appropriate restriction endonucleases, iso lated characterized and used for various purposes.

Gene Library: By the combination of restriction enzymes and various cloning vectors the entire DNA of an organism can be packed into a suitable vector.

DNA Probes: Probes are used to search libraries for specific genes or cDNA molecules. Gene libraries contain large numbers of DNA fragments and it is extremely difficult to find out or choose a specific DNA sequence of interest from these.

Forms of Gene Therapy:

i) Germ - line gene therapy: Would allow transmission of the modified genetic information to the next generation and is not currently accepted as an appropriate therapeutic approach. For ethical reasons, opposition to germ-line gene therapy is likely to continue in the forescable future.

ii) ***Somatic gene therapy:*** refers to modification of the so- matic, differentiated cells of the body. It is used in an effort to correct inherited diseases such as cystic fibrosis and acquired disorders such as rheumatoid arthritis or malig- nancies.

A growing use of gene therapy involves transient gene ex- pression to treat a variety of diseases.

Gene therapy is now being considered or used for:- i) Killing cancer cells ii) Providing chromoprotection to normal cells. iii) Preventing coronary restenosis or enhancing vascu- larization. iv) Providing DNA - based immunization (e.g. viral DNA), in which the injection of DNA results in antigen ex- pression and generation of an immune response; and v) impairing viral replication.

Somatic Cell Gene Therapy: Two approaches can be used to de- liver genes to somatic cells.

Ex vivo gene therapy: involves the removal of cells from patient, the introduction of therapeutic gene(s) using viral or non-viral vectors, and the autologous transplantation of the gene-corrected cells into the patient.

In vivo gene: therapy refers to the direct delivery of the therapeutic gene to the target sites of pathology of the diseased individual (e.g. IV infusion, stereotaxic infection).

Gene transfer vectors: A vector or vehicle is required to transfer a gene to an appropriate cell. Two major classes of vectors are used for transferring nucleic acids into cells for the purposes of gene therapy:-

i) Viral and and ii) Non viral vectors

i) ***Viral vectors:*** have been genetically engineered so that the viruses transfer exogenous (therapeutic) nucleic acids into cells through a process called as transduction. These viruses include:- Retroviruses (RV), Adenovirus (AV), Lenti virus (LV), Adeno- associated virus (AAV) *8* Herpes simplex virus (HSV).

ii) Non viral vectors: Consist of nucleic acids that are typically complexed with other chemicals to facilitate gene transfer. Al- though non-viral vectors offer improved safety by avoiding viral components, their gene transfer efficiencies are generally much lower than those of viral vectors.

APPLICATIONS OF RECOMBINANT DNA TECHNOLOGY

Recombinant DNA technology (genetic engineering) has revolutionized the application of molecular biology to medical/ agricultural sciences that has immensely benefitted the mankind.

A few of the useful practical applications of recombinant DNA technology are listed below:-

1. **Gene Mapping Localized Specific**
   Genes to Distinct Chromosomes: Somatic cell hybridization and *in situ* hybridization are two techniques used to accomplish localising gene.

Table: Localization of human genes.

Gene	Chromosome	Disease
Insulin	11p15	
Prolactin	6p23-q12	
Growth hormone	17q21-qter	Growth hormone deficiency
α-Globin	16p12-pter	α-Thalassemia
β-Globin	11P12	β-Thalassemia, Sickle cell
Adenosine deaminase	20q13-qter	Adenosine deaminase deficiency
Phenylalanine hydroxylase	12q24	Phenylketonaria
Hypoxanthine-guanine phosphoribosyl transferase	Xq26-q27	Lesch-Nyhen syndrome
DNA segment G8	4p	Huntigton's chorea

Table: Examples of Genetically Engineered Recombinant Proteins used in Medicine

Recombinant Proteins	Clinical Applications
I. Hormones growth factors	(The efficiency an approval of some of these agents remain and under investigation)
◆ Growth hormone (GH)	Growth hormone deficiency
◆ Insulin - like growth factor 1 (IGF-1)	Growth disorders resistant to growth hormone.
◆ Insulin	Diabetes Mellitus
◆ Follicle stimulating hormone (FSH)	Ovulation induction.
◆ Luteinizing hormone (LH)	Ovulation induction.

2. ***Proteins can be produced for Research & Diagnosis:*** Examples of Recombinant Proteins are listed in the following table.
3. ***AIDS Test:*** By using recombinant DNA techniques, the diagnosis like AIDs by laboratory has become simple and rapid.
4. ***Diagnosis of molecular diseases:*** Many genetic diseases that yield developmental abnormalities can be detected by characteristic patterns in DNA primary structure. Such mutational changes in DNA sequences are identified by restriction fragments analysis and Southern blotting, using appropriate DNA probes. Analysis of this type could be done in understanding the molecular basis of diseases like sickle cell anaemia, thalassemia, familial hypercholesterolemia, cystic fibrosis etc.
5. ***Prenatal diagnosis:*** If the genetic lesion is understood and a specific probe is available prenatal diagnosis is possible. DNA from cells collected from as little as 10 ml of amniotic fluid or by chorionic villi biopsy can be analyzed by Southern blot transfer.
6. ***Application in forensic medicine:*** Advances in genetic engineering have greatly helped to specifically identify criminals and settle the disputes of parenthood of children. The restriction analysis pattern of DNA of one individual will be very specific (DNA finger printing), but the pattern will be different from person to person.
7. ***Transgenesis:*** The somatic gene replacement therapy will not pass on to the offspring. Transgenesis refers to the transfer of genes into fertilized ovum which will be found in somatic as well as germ cells and passed on to the successive generations.

Note: The above depicts the brighter aspects of genetic engineering. But it is to be remembered that there are likely "hazards" also e.g. by any change, in the course of cloning using bacterial vectors, some drug- resistant or harmful bacteria are produced and released, the new varieties may cause incurable diseases to the humanity and the live-stock. Scientists should use the technology for useful purposes only like atomic energy and not for harmful purposes to humanity.

8. *Pedigree analysis:*
9. ***Polymerase chain reaction:*** It is a method of amplying a target sequence of DNA. The PCR is used (1) to detect infectious agents (2) to make prenatal genetic diagnoses (3) to detect allelic poly- morphisms; (4) to study evolution; and (5) to establish precise tissue types for transplants.
10. ***Gene therapy:*** Gene therapy is generally defined as the delivery of nucleic acids to alter or prevent a pathologic process.

Table: Examples of Disorders Potentially Amenable to Gene Therapy

Disease	**Gene Therapy Strategy**
I. In ted diorders	**Gene Addition**
◆ Cystic Fibrosis	Express CFTR [Cystic fibrosis transmembrane regulator] in pulmonary system and / or GI tract.
◆ Familial hypercholesterolemia	Express LDL-receptor in liver
◆ Hemophilias A and B	Express Factor VIII or IX and secrete in circulation.
◆ Thalassemia p	Express normal globin in RBCs.
◆ Immunodeficiencies	Express mutant genes, such as adenosine deaminase.
◆ Metabolic disorders	Express missing enzymes or transporters.
◆ Duchenne's muscular dystrophy	Express mutant dystrophin protein muscle cells.
◆ Retinitis Pigmentosa [recessive]	Express normal protein in retina.
	Gene Correction
◆ Lesch - Nyhan	Modify hypoxantine phosphoribosyl transferase locus.
◆ Retinitis pigmentosa (dominant)	Correct missense mutation.
◆ Sickle cell disease	Correct β globin mutation.
◆ Cystie fibrosis	Correct θ F 508 mutation in pulmonary system.
II. Cardiovascular diseases	**Modify vascular biology**
◆ Coronary Artery Stenosis	Block cell proliferation in vessel wall.
◆ Peripheral vascular disease	Induce angiogenesis.
◆ Hypertension	Express genes (e.g. tissue kallikrein) to induce vasodilation)
III. Cancer	**Multiple Approaches**
◆ Many types	Express immunostimulants in or near malignant cells. Express toxic genes in tumor cells (e.g. herpes simplex virus TK) Express genes in normal cells to protect from chemotherapy.
IV. Infectious diseases	
◆ Viral diseases	Express genes that block viral replication or function, including ribozymes, decoys, dominant negative proteins.
◆ Many	Express antigens as recombinant vaccines.
V. Miscellaneous	
Rheumatoid arthiritis	Express anti-inflammatory cytokines in joints.
Parkinson's disease	Express gene required for L-dops synthesis instriatum.
Neurodegenerative disease	Express neurotrophic factors.

References

1. Edward H. Schuchman, Janet E. Carter, Robert J. Desnick; Gene therapy strategies for the treatment of neurodegenerative and other genetic dis- eases: Principles and Practice of Medical Genetics; 4th ed. 1: 875-92.
2. Gordon R. Bernard et al. (2001); Efficacy and Safety of Recombinant Human Activated Protein C for severe sepsis; NEJM: 344: 699 – 707.
3. Lewin B. (1998): Genes VI Oxford Univ. Press.
4. Lynda C' Schneider et al. (1998); Long term therapy with recombinant interferon gamma for atopic dermatitis; Ann Allergy Asthma Immunol: 80: 263 - 8.
5. Mark A. Kay, David W. Russell; Gene therapy: Harrison's principles of Internal Medicine. 15th ed; 1: 412 - 18.
6. Msrtin J.B. Gussella J.F. (1986); Huntington's disease; Pathogenesis and management; NEJM: 315: 1267.
7. Ssmbrook J. et al. (1989); Molecular cloning: A Laboratory Manual Cold Spring Harbor Laboratory Press.
8. Spector D.L. et al. (1998); Cells: A Laboratory Manual, Cold spring Harbor Laboratory Press.
9. Watson J.D. et al. (1992); Recombinant DNA, 2nd ed. Scientific American Book Freeman.
10. Weather D.J. (1991): The New Genetic *8* Clinical Practice, 3rd ed. Oxford Univ. Press.

IRRITABLE BOWEL SYNDROME-CURRENT CONCEPTS

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Irritable bowel syndrome (IBS) is one of the most common functional gastrointestinal disorder encountered worldwide. This bowel disorder comprises of abdominal pain associated with defecation or a change in bowel habit with features of altered defecation and distension with no demonstrable abnormality. The diagnosis is based on clinical findings and the exclusion of other disorders. The treatment of IBS still involves a comprehensive, multidisciplinary approach that includes medical management of dominant symptoms, dietary modifications and, possibly, psychotherapy.

Depnition

Since mid-1970's there have been significant gains made in our understanding of IBS. We have witnessed a progression from attributing IBS to psychosocial issues to understanding that IBS is a disorder of visceral hypersensitivity, abnormal gut motility and complex "brain-gut" interactions.

The irritable bowel syndrome is defined on the basis of the recently modified Rome criteria as the presence for at least 12 weeks (not necessarily consecutive) in the preceding 12 months of abdominal discomfort or pain that cannot be explained by structural or biochemical abnormalities and that has at least 2 of the following 3 features: pain is relieved with defecation, its onset is associated with a change in the frequency of bowel movements (diarrhea or constipation), or its onset is associated with a change in the form of the stool (loose, watery, or pellet-like).'

Visceral hypersensitivity in IBS patients has been well studied and it appears that balloon inflated in rectum of IBS patient is perceived painful at much lower threshold than in normal patient, a concept known as allodynia . Previous studies have demonstrated that patients with IBS have discrete clustered contractions in the small bowel associated with pain', while other patients with IBS have prolonged contraction within the colon, especially postprandially . Recently, attention has focused on the "brain-gut" interactions in the IBS patient.

Epidemiology

IBS remains one of the most common medical conditions diagnosed in primary care practice today. In the western world, IBS accounts for nearly 12% of primary care patient visits and approximately 28% of referrals to gastroenterologist. For many, IBS is a disorder that affects all aspects of life and significantly reduces the quality of life. Recent European and American epidemiologic investigations do validate that there is a high prevalence of IBS, especially in women aged 35-44 years and also confirm significant impact of this disorder on healthcare utilization, functional status and quality of life. This recent data also highlights the current lack of effective treatment, and points to the need for newer treatments for this common ailment.[5,6,7,8]

Pathophysiolgic Features

Altered bowel motility, visceral hypersensitivity, psychosocial factors, an imbalance in neurotransmitters, and infection have been proposed to play a role in IBS.

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1. Altered Bowel Motility
   Over the past 50 years, alterations in motility of the colon and small bowel have been described in patients with the IBS. Psychological or physical stress' and ingestion of food' may alter the contractility of the colon. Abnormal motility of the small intestine during fasting, such as loss of the migrating motor complex" and the presence ofboth discrete, clustered contractions and prolonged, propagated contractions,' has been described in patients with the irritable bowel syndrome.
2. Visceral Hypersensitivity
   Balloon-distention studies of the rectosigmoid have shown that patients with the IBS experience pain and bloating at balloon volumes and pressures that are significantly lower than those that induce pain in control subjects, a phenomenon referred to as visceral hypersensitivity. This hypersensitivity may exist at the enteral or neural level either in afferent or efferent pathways, especially in neurogenic areas rich in catecholamines and serotonin.
3. Psychosocial Factors
   Psychological stress can alter motor function in the small bowel' and colon", both in normal subjects and in patients with the irritable bowel syndrome. Up to 60 percent of patients have psychiatric symptoms such as somatization, depression, and anxiety, and patients with a diagnosis of the IBS are more likely to have these symptoms than are persons who have never sought medical care for bowel problems.'," The presence or absence of a history of abuse in childhood (sexual, physical, or both) is correlated with symptom severity in IBS patients.'
4. *Neurotransmitter Imbalance*
   Recent studies show that neurotransmitters are involved in the pathogenesis of the IBS. About 95% Serotonin is in the gastrointestinal tract, within entero cells, neurons, mast cells, and smooth-muscle cells. When released by enterochromafinee cells, serotonin results in such physiological responses as intestinal secretion and the peristaltic reflex and in such symptoms as nausea, vomiting, abdominal pain, and bloating.' Preliminary evidence suggests that patients with IBS have increased serotonin levels in plasma and in the rectosigmoid colon."'
5. Infection and Inflammation
   There is evidence that inflammation of the enteric mucosa or neural plexuses initiates or contributes to symptoms associated with IBS.

Diagnosis

In clinical practice, diagnosis is based on clinical symptoms and limited diagnostic screen, taking into account warning features suggestive of organic disease.

After a complete history has been obtained, all patients with lower gastrointestinal tract symptoms should undergo a complete physical examination and laboratory testing, including a complete blood count, blood-chemistry tests, liver-function tests, and measurement of thyrotropin. The diagnosis of the IBS is suggested when patient's symptoms meet the Rome criteria. In the majority of cases, there are no abnormalities on physical examination or laboratory testing and there are no findings suggestive of a structural disorder. Therefore, the IBS can be reasonably diagnosed on the basis of flexible sigmoidoscopy alone, in patients who are less than 50 years old, or colonoscopy, in those who are 50 or older; barium enema and flexible sigmoidoscopy are often substituted for colonoscopy. In patients with diarrhea, a biopsy specimen should be obtained from the mucosa of the descending colon to rule out microscopic colitis.

Any abnormalities on physical examination or laboratory testing or if an alarm symptom is present, further evaluation with tests, such as colonoscopy, CT Scan of the abdomen and pelvis, and radiology of the small intestine. Alarm symptoms include gastrointestinal bleeding such as occult blood in the stool, hematochezia, or anemia; anorexia or weight loss; fever; persistent diarrhea causing dehydration; severe constipation or fecal impaction; a family history of gastrointestinal cancer, inflammatory bowel disease, or celiac sprue; and the onset of symptoms at the age of at least 50 years. In 1985, the American College of Physicians recommended the use of alarm symptoms to guide treatment

Treutment

When the irritable bowel syndrome is diagnosed the establishment of trust in the physician – patient relationship should be given a high priority in order to maximize the efficacy of treatment and minimize "doctor shopping."

A diary of food intake and symptoms can be useful in identifying foods that may be associated with symptoms of the IBS. Some patients benefit from avoiding or limiting their intake of caffeine, alcohol, fatty foods, gas-producing vegetables, or products containing sorbitol, such as sugarless gum and dietetic candy. The avoidance of constipating foods and the addition of 20 to 30 g of fiber per day either in the diet or in the form of supplements such as bran, polycarbophil, or a psyllium derivative may help relieveconstipation[24,25,26,27]

A rational approach to treating the IBS uses the patient's symptoms as a guide. For patients in whom the disorder is manifested predominantly as abdominal pain, a variety of medications have been used. Antispasmodic agents may reduce abdominal pain or bloating, in refractory cases, nitrates are occasionally useful for direct relaxation of smooth muscles. A recent meta-analysis showed effectiveness of antispasmodic agents and tricyclic compounds in treating selected patients with the irritable bowel syndrome. ' Small studies have shown that tricyclic compounds in low doses relieve unexplained abdominal pain. Side effects like sedation, dry mouth and eyes, and weight gain limit the use of primary tricyclic amines. Secondary tricclic amines such as nortriptyline and desipramine may be less likely to have side effects.

Possible role of Serotonin in the pathogenesis of the IBS led to development of two new agents – alosetron, a 5-HT~ – receptor antagonist, and tegaserod, a 5-HT4 agonist – have been shown to diminish visceral sensitivity to rectal distention in women who have diarrhea as the predominant symptom of the irritable bowel spdrome and in those who have constipation as the predominant symptom, respectively.[31,32]

The patients with diarrhea-predominant irritable bowel syndrome, antidiarrheal agents such as loperamide" and diphenoxylate may help decrease the frequency of bowel movements and improve the consistency of stool. In a study of women with this form of the irritable bowel syndrome, alosetron prolonged colonic transit, reduced the frequency of bowel movements and the urge to defecate, improved the consistency of stool, and decreased abdominal pain. However, this drug has been removed from the market because of side effects such as severe constipation, ischemic colitis, and bowel perforation. In uncontrolled diarrhea, cholestyramine has been used to bind bile acids that may be responsible for increased secretion and decreased absorption of water in the colon. In refractory cases, a short course of antibiotics may reduce the diarrhea, presumably by altering the intestinal flora.

The patients with constipation-predominant irritable bowel syndrome, high-fiber diet may alleviate constipation and related symptoms such as abdominal pain and tenesmus. Constipation can also be safely treated with osmotic laxatives such as nonabsorbable carbohydrates (lactulose and sorbitol), milk of magnesia or magnesium citrate, or a polyethylene glycol solution. Two new classes of compounds, aminoguanidine indoles such as tegaserod and benzofurans such as prucalopride, act specifically on 5-HT4 receptors. These agents shorten the transit time in the colon and small intestine, increase the frequency of bowel movements, and increase the softness of stools.. Tegaserod is the one recently approved by FDA in this group of patients.

Summary

The interaction between psychosocial factors and the genesis of all forms of the irritable bowel syndrome is poorly understood. The most provocative observations in this respect are that persons with the syndrome who seek care are those in whom the syndrome is accompanied by a psychiatric disorder and that the syndrome may develop in patients who have both infectious gastroenteritis and a psychiatric disorder. The potential benefits of supportive therapy, relaxation exercises, hypnosis, cognitive behavioral therapy, and psychodynamic interpersonal psychotherapy are well recognized."

The irritable bowel syndrome is a common disorder that has a pronounced effect on the quality of life and that accounts for a large proportion of health care costs. Common pitfalls in diagnosing and treating this disorder include unnecessary repetition of tests, More to establish trust in the physician – patient relationship, and failure to provide the patient with realistic expectations regarding the efficacy of medications. A concise diagnostic evaluation and prompt institution of symptom-guided therapy can help alleviate the pain and suffering experienced by patients with the irritable bowel syndrome.

References:

1. Thompson WG, Longstreth G, Drossman DA. Functional bowel disorders and functional abdominal pain. In: Drossman DA, Corazziari E, Talley N, Thompson WG, Whitehead W, eds. The functional gastrointestinal disorders: diagnosis, pathophysiology, and treatment. 2nd ed. MeLean, Va.: Degnon, 2000:351-75.
2. Whitehead WE, Holtkotter B, Enck P, I-Ioelzl k Holmes KD, Anihonv 3. Shnb~in HS, Schuster MM.: Tolerance for rectosigmoid distention in irritable bowel syndrome. Gastroenterology. 1990 May;98(5 Pt 1):1187-92,
3. Kellow JE, Phillips SF Altered small bowel motility in irritable bowel syndrome is correlated with symptoms. Gastroenterology. 1987 Jun;92(6): 1885-93.
4. Chey%Y, Jin HO, Lee MH"Sun SW, l.,ee kY. Colonic motility abnormality in patients with irritable bowel syndrome exhibiting abdominal pain and diarrhea. Am J Gastroenterol. 2001 May;96(5):1499-506.
5. Sandler RS Epidemiology of irritable bowel syndrome in the United States. Gastroenterology. 1990 Aug;99(2):409-15.
6. Hungin APS, Tack J, Whorwell P J, Mearin F. The medical and societal impact of Irritable bowel syndrome (IBS):the truth in IBS (T-IBS) survey. Poster presentation at World Congress of Gastroenterology, Bangkok, February 2002
7. Hungin APS, Tack J, Whorwell PJ, Dennis E, Barghout V. Irritable bowel syndrome (IBS): prevalence and impact in the USA-The truth in IBS (T-IBS) survey. Poster presentation at the Ameican College of Gastroenterology 67 Annual Scientific Meeting, Seattle, October 2002.
8. Hungin APS, Tack J, Whorwell P J, Dennis E, Barghout V. The truth in IBS (T- IBS) survey- healthcare utilization and medication use among IBS patients in the USA. Poster presentation at the American College of Gastroenterology 67 Annual Scientific Meeting, Seatlle, October 2002.
9. Almy TP. Experimental studies on the irritable colon. Am J Med 1951;10:60-67.
10. Snape WJ Jr, Carlson GM, Matarazzo SA, Cohen S. Evidence that abnormal myoelectrical activity produces colonic motor

dysfunction in the irritable bowel syndrome. Gastroenterology 1977;72: 383-387

11. Kumar D, Wingate DL. The irritable bowel syndrome: a paroxysmal motor disorder. Lancet 1985;2:973-977
12. Kellow JE, Phillips SF. Altered small bowel motility in irritable bowel syndrome is correlated with symptoms. Gastroenterology 1987;92: 1885-1893.
13. Almy TP, Kern F Jr, Tulin M. Alterations in colonic function in man under stress. II. Experimental production of sigmoid spasm in healthy persons. Gastroenterology 1949;12: 425-436
14. Drossman DA, McKee DC, Sandler RS, et al. Psychosocial factors in the irritable bowel syndrome: a multivariate study of patients and nonpatients with irritable bowel syndrome. Gastroenterology 1988;95:701-708
15. Whitehead WE, Bosmajian L, Zonderman AB, Costa PT Jr, Schuster MM. Symptoms of psychologic distress associated with irritable bowel syndrome: comparison of community and medical clinic samples. Gastroenterology 1988;95: 709-714.
16. Drossman DA. Sexual and physical abuse and gastrointestinal illness. Scand J Gastroenterol Suppl 1995;208:90-96.
17. Gershon MD. Roles played by 5-hydroxytryptamine in the physiology of the bowel. Aliment Pharmacol Ther 1999;13: Suppl 2:15-30.
18. Bearcroft CP, Perrett D, Farthing MJG. Postprandial plasma 5-hydroxytryptamine in diarrhoea predominant irritable bowel syndrome: a pilot study. Gut 1998;42: 42-46
19. Bose M, Nickols C, Feakins R, Farthing MJ. 5-Hydroxytryptamine and eaterochromaf6n cells in the irritable bowel syndrome. Gastroenterology 2000;118: Suppl: A563-A563. abstract
20. Stewart GT. Post-dysenteric colitis. BMJ 1950;1:405-409.
21. Chaudhary NA, Truelove SC. The irritable colon syndrome: a study of the clinical features, predisposing causes, and prognosis in 130 cases. Q J Med 1962;31:307- 322.
22. McKendrick MW, Read NW. Irritable bowel syndrome – post salmonella infection. J Infect 1994;29: 1-3.
23. Health and Public Policy Committee, American College of Physicians. Endoscopy in the evaluation of dyspepsia. Ann Intern Med 1985;102:266-269..
24. Cann PA, Read N%, Holdsworth CD, What is the benefit of coarse wheat bran in patients with irritable bowel syndrome? Gut 1984;25:168-173
25. Toskes PP, Connery KL, Ritchey TW. Calcium polycarbophil compared with placebo in irritable bowel syndrome. Aliment Pharmacol Ther 1993;7:87-92.
26. Jalihal A, Kurian G. Ispaghula therapy in irritable bowel syndrome: improvement in overall well-being is related to reduction in bowel dissatisfaction. J Gastroenterol Hepatol 1990;5:507-513.
27. Prior A, Whorwell P J. Double blind study of ispaghula in irritable bowel syndrome. Gut 1987;28:1S10-1513.
28. Jailwala J, Imperiale TF, Kroenke K. Pharmacologic treatment of the irritable bowel syndrome: a systematic review of randomized, controlled trials. Ann Intern Med 2000;133:136-147.
29. Rajagopalan M, Kurian 6, Jacob J. Symptom relief with amitriptyline in the irritable bowel syndrome. J Gastroenterol Hepatol 1998;13:738-741.
30. Clouse RE. Psychotropic medications for the treatment of functional gastrointestinal disorders. Clin Perspect Gastroenterol 1999;2:348-56.
31. Miura M, Lawson DC, Clary EM, Mangel AW, Pappas TN. Central modulation of rectal distension-induced blood pressure changes by alosetron, a 5-HT3 receptor antagonist. Dig Dis Sci 1999;44:20-24.
32. Schikowski A, Mathis C, Thewissen M, Ross H-G, Pak MA, Enck P. Dose- dependent modulation of rectal afferent sensitivity by a 5-HT4 receptor agonist. Gastroenterology 1999;116: A643-A643.abstract
33. Cann PA, Read NW, Holdsworth CD, Barends D. Role of loperamide and placebo in mmagement of irritable bowel syndrome (IBS). Dig Dis Sci 1984;29:239-247
34. Camilleri M, Northcutt AR, Kong S, Dukes GE, McSorley D, Mangel AW. EKcacy and safety of alosetron in women with irritable bowel syndrome: a randomised, placebo-controlled trial. Lancet 2000;355:1035-1040.
35. Sciarretta G, Fagioli G, Furno A, et al. 75Se HCAT test in the detection ofbile acid malabsorption in functional diarrhoea and its correlation with small bowel transit. Gut 19&7;28:970-975.
36. Pimentel M, Chow EJ, Lin HC. Eradication of small intestinal bacterial overgrowth reduces symptoms of irritable bowel syndrome. Am J Gastroenterol 2000;95:3503-3506.
37. Prather CM, Camilleri M, Zinsmeister AR, McKinzie S, Thomforde G. Tegaserod accelerates orocecal transit in patients with constipation-predominant irritable bowel syndrome. Gastroenterology 2000;118:463-468
38. Drossman DA, Thompson WG. The irritable bowel syndrome: review and a graduated multicomponent treatment approach. Ann Intern Med 1992;116: 1009- 1016 .

RECURRENT PREGNANCY LOSS / RECURRENT ABORTION/ RECURRENT MISCARRIAGE/HABITUAL ABORTION

Tabassum Parveiz M.D.

Definition:

RPL is defined as 3 or more consecutive spontaneous abortions before 20 wks of gestation or a fetal weight less than 500 gms.

◆ 2 Types – Primary Aborters: Women without a previous liveborn infant

Secondary Aborters: Women with atleast one prior liveborn infant

Incidence: 0.5 – 1% of all pregnancies.

Recurrence Risk:

The reproductive H/o patient is very important as relative risk of abortion increases with no. of previous pregnancy failures.

No. of Prior losses	% Risk of loss in next pregnancy
Atleast one live born infant	
0	12 %
1	24 %
2	26 %
3	32 %
4	26 %
No live born infant	
2 or more	40 – 45 %

Etiology: In 50 % cases a Probable cause is identified

1. Genetic factors: (5%)

A) Chromosomal abnormalities of conceptus

◆ Are a common cause of early sporadic abortion [single losses] but not a major cause of RPL

◆ Responsible for 50-60% of 1st TM abortion.

◆ Most common are aneuploidies, which are present in 80% of blighted ova and 5-10% abortion in which fetus are present.

◆ Are detected by examination of POC.

◆ Occur due to errors of maternal or paternal meiosis I or II, super fecundation of an egg by 2 sperms of form a chromosome division in absence of cytoplasmic division with advancing maternal age or due to fertilization of ageing ova or sperm.

i) Autosomal trisomies (40 – 50%)
   ☐ Includes 13, 16* 18, 21 and 22.
ii) Monosomy X – (20-25%)
   ☐ Have a karyotype 45 X.
   ☐ If pregnancy is not aborted : identified at birth as individual with Turner's syndrom,
iii) Polyploidy – Triploidy (16%)
   - Mostly seen in IVF conceptuses
   - Triploid fetus may result in formation of V. mole.

B) Parental chromosomal abnormalities (3-5%)

- Is higher in couples who have not experienced a live birth.
- The structural re-arrangements could be de-nova. Occurring during gametogenesis or could be inherited from one of the parents who is a carrier for these abnormalities.

Includes:

◆ Translocations – 2/3rd reciprocal balanced translocation
   - 1/3rd Robertsonian translocation
   - Translocation carriars are phenotypically normal.

◆ Others –
   - Inversions
   - Mosaicism

II. Anatomic abnormalities of uterus (10-15%)

- Usually responsible for 2nd Tm abortion
- May be congenital or acquired

A) Congenital –

a) Mullerian duct abnormalities –

1. Unicornuate uterus
2. Bicornuate uterus
3. Septate uterus
4. Didelphic uterus

b) DES exposure in utero –

Can cause multiple uterine anomalies most common hypoplasia contributing to 1st and 2nd Tm spontenous abortion, incompetent cervix and prematiure labour.

c) Uterine artery anomalies –

B) Acquired –

1. Cervical incompetence –
   - May be congenital (rare) or acquired (iatrogenic)

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- Usually late 2nd Tm abortion.
- Caused by – cervical trauma
- Following D&C, Vaginal operative delivery through an undilated cervix, amputation of cervix, cone biopsy.

2. Intrauterine adhesions (synechiae) –
   - Result from intrauterine infection combined with surgical trauma like – postabortal or postpartum curettage for retained POC.
   - Tubercular endometritis.
3. Submucus fibroids –

Less common causes includes

4. Endometriosis
5. Retroverted uterus

III. Endocrine abnormalities (10 –15%) –

a) Luteal phase deficiency –

In adequate endometrial maturation due to insufficient progesteron – early pregnancy loss.

◆ There is much controversy about LPD as a cause of RPL.

◆ Decreased progesteron level is the effect rather than cause of abortion.

b) Polycystic ovarian syndrome – (PCOS)

◆ In RPL, 82% pt. Shows a evidence of PCOS.

◆ Hypersecretion of LH, may lead to

1. Direct inhibition of oocyte maturation inhibitor (omi) leading to production of physiologically aged oocyte.
2. Altered synthesis of prostaglandins.
3. Increased androgen systhesis by theca cells.
4. Production of abnormal glycoform of LH causing abnormal signal transduction and anovulation.

c) Thyroid deficiency –

◆ Unlikely cause of RPL.

d) Diabetes –

Poorly control diabetes with elevated blood sugar and glycosylated Hb in first Tm.

e) Androgen and Prolectin disorders –

can lead to corpus leuteum dysfunction.

IV. Infections – (5%)

◆ More likely to cause sporadic loss.

◆ Uterine or disseminated.

List includes –

Syphilis, Rubella, Herpes, Cytomegalovirus, Listeria, Toxoplasmosis, Ureaplasma, Brucella, Mycoplasma, E.coli, Tubercular bacilli.

V. Immunological factors – (20 – 40%)

a) ***Abnormal cellular immuno response*** – (Embryo/trophoblast toxic factors/cytokines)

Trophoblast antigens —— + —-à macrophages and lymphocytes —-à abnormal cellular response by TH1 cells ——à release of cytokins, IFN – g and TNF -à inhibit in-vitro development of embryo.

b) Abnormal humoral immune response.

1. Autoimmune factors

Include *antiphospholipid antibody syndrome* (APAS), SLE.

◆ Antibodies are formed against negatively charged phospholipids. In my recent case study of 50 pts of recurrent abortions in 2001-2002 the autoimmune factor of antiphospholipid antibody formed against negatively charged phospholipid has been demonstrated in 4 patients. Its occurrence would be surely demonstrated in more pts of recurrent abortions. If all such patients are thoroughly screened for these autoimmune factors.

Mainly 2 types – LA & ACA.

MOA – Increased thromoboxane and decreased prostacycline synthesis, reduced anti thrombin III synthesis and protine C deficency.

◆ APA can inhibit in vitro syncytial trophoblast formation.

2. Allo – immune factors –

◆ Active immune modulation of maternal system protects the embryo from rejection.

◆ Circulating factors proposed as being immuno suppresive include blocking antibodies, early pregnancy factor IL-2, local suppressor cells & certain cytokines.

VI. Miscellaneous factors – (10%)

i) Environmental toxins – heavy metal toxicity, prolonged exposure to organic solvents.

ii) Drugs – Antiprogestogens, Antineoplastic agents, inhalation anaesthetics, Nicotine and ethanol.

iii) Ionizing radiation.

iv) Medical illnesses – Cardiac, renal, Hematological, Wilson's disease, Crohn's disease.

v) Coitus

vi) Exercise

vii) Incompatible ABO group matings.

VII. Idiopathic – (40 –50%)

Management –

History to be elicited in between the pregnancies –

From the female partner –

Age, Menstrual history, duration of marriage contraception use, no. of previous pregnancies and their outcome, gestational age at outcome. Type of abortions associated with pain, post-abortal curretage, histopath confirmation of POC, karyotype of abortions.

Past Medical History:

1. Diabetes Mellitus
2. Thyroid
3. T.B
4. Chronic renal disease
5. TORCH infection

Past surgical history: On genital tract

Family History:

1. Chromosomal disorders
2. Congenital anomalies
3. Diabetes mellitus

History of subtance abuse:

From the husband –

Age, H/o previous successful proven fertility, occupation, medical illness, testicular trauma, family H/o – Chronic disease

- Oligo/Polyspermia
- Substance abuse
- H/o consanguinity

History during Pregnancy:

LMP, precious cycles, use of oral contraceptive, use of ovulation inducing agents.

Bleeding/pain/pelvic pressure.

On examination:

- Mental status
- General examination
- Vital signs
- Stigma of endocrinal / systemic disease
- Systemic examination.

P/S
- Cervix / vaginal infection
- Condition of ext. OS.

P/V
- Uterus
  - size, shape, mobility, tenderness
  - any anomalies
  - adenexa
  - cervical incompitence/tears

Investigations:

1. Genetic:

- Parental peripheral blood karyotyping with banding techniques.
- Karyotyping of abortus/amniotic fluid/amnion/ placenta.

2. Antiphospholipid antibody syndrome-

- Plateletcount, B.T., C.T.
- To detect LA
  - Activated partial thromboplstin time. (APTT)
  - Kaolin clotting time (KCT)
  - Dilute Russel viper venom time. * (d RVVT)
  - Tissue thromboplastin inhibition time (TTIT)
  - To detect ACA
  - A standerdised ELISA is used for both IgG & IgM classes of ACA

3. Luteal Phase Defect

- *Endometrial biopsy* – Ideally 10 days after LH surge. In out of phase biopsies – Serum androgen & prolactin profile.
- *Serum progestron assay* – Sum of three values obtained 3 days apart between 4 & 11 days before the next expected menses. Value < 15ngm /ml is significant.

4. Hypothyrodism –

- TFT including T3, T4 & TSH.
- Detection of antithyroid antibody.

5. Diabetes –

- Blood sugar profile.
- Glycosylated Hb estimation.

6. PCOS –

- Estimation of LH, FSH : LH.
- USG – Most sensitive.
- Laparoscopy.

R
M

7. Infections –

- Complete hemogram
- TORCH titre
- ELISA
- Cervical swab culture
- ESR
- VDRL
- Urine

8. Uterine Anomalies-

- Clinical examination
- HSG
- Laproscopy
- USG
- Sono-hystrography
- Hystroscopy

9. Cervical incompitence-

- Interconceptional period -
  - Eaxy passage of No. 8 Hegar's dilator.
  - Premenstrual hystro - cervicography
- During pregnancy-
  - P/V examination.
  - On USG: cervical length < 2.5 cm
  - Diamter of int. os > 23 mm.

10. Endometriosis-

- USG
- Laproscopy

Treatment

1. Genetic

- No specific therapy
- Genetic counselling
- Donor sperm/Donor eggs.
- Gene treapy
- The couple may opt. Or adoption.

2. Anatomic abnormalities –

- Uterine septum – Transcervical lysis
- Broad uterine septum – Modified Jone's metroplasty.
- Tompkin's metroplasty
- Bicornuate & didelphic uterus – Strassman's metroplasty.
- Intrauterine adhesions – hystroscopic resection.
- Submucous fibroids – myomectomy.
- Cervical incompitence – interval tracheloraphy
  - during pregnancy – Macdonald's stitch
  - Shirodkar's stitch
- Endometriosis –

- Mild – no treatment, analgesics for pain.
- Modarate to severe – Combined OC's X 6 months.
  Danazole 200-800mg/day X 6 months
  Oral MPA – 30mg/day X 6 months.
  Parentral MPA – 100 mg i/m every 2 wks. For 3 months followed by 200 mg i/m monthly for 3-6 months.

◆ Surgical –

◆ Laproscopic adhesiolysis, Diathermies, Laser vaporization by CO2 or Nd/Yag laser, excision of cysts, presacral neurectomy.

3. **LPD** –

◆ Progestron supplementation (started 3 days after ovulation)

- Vaginal suppositories –25 – 50 mg BD (till 8-9 wks of gestation)
- I/m 12.5 mg/day (till 8-9 wks of gestation)
- or as soon as preg. Diagnosed weekly i/m inj. Of 17 - hydroxy progestron caproate (maintance, proluton depot, procaprin) – 250 – 500 mg X 16 weeks.

◆ HCG – After LH surge 2000 – 5000 IU every 2 – 5 days X 12 wks. I/m (Pubergen, Ovidac, Fertigyn, Life)

4. **PCOS** –

◆ Wt. Loss.

◆ 6 months of OC's.

◆ Dexamethasone 0.5 mg or prednisolone 5 mg at bed time.

◆ LHRH agonists.

◆ Surgery – if medical therapy fails or hyperstimulation occurs

◆ Laproscopic multiple puncture of the cysts with electrocuatry or laser.

5. **Hypothyroidsm – Eltroxin**
6. **Diabetes – Counselling, insulin.**
7. **Infections** – Treated by appropriate antibiotics to both the partners.
8. **Anti Phospholipid antibody syndrome** –

◆ Low dose aspirin – 75 – 80 mg/day or low dose aspirin 75 – 80 mg/day + deparin 5000 – 10000 U X BD S/c

◆ Treatment is started as soon as pregnancy test is positive and continued till 32 wks.

◆ Monitoring is done by –

- Levels of APA – both LA & CA monthly.
- Platelet count every 2 wks.
- Serial bone minral density in women receiving heparin.
- USG and Doppler monitoring of fetus.

◆ IVIG – can be used to treat APA syndrome with or without heparin. Dose – 0.5 mg/Kg/month beginning before conception followed by 500 – 600 mg/Kg at 5 wks, followed by 300 – 400 mg/Kg every 3 weeks till 22 wks.

9. **Allommune factors** –

◆ Immunisation of female partner by leukocytes from–

- Male partner
- Seminal plasma
- Third party leukocytes
- Trophoblastic preparation

These may suppress CMI response and NK activity.

◆ In non responders passive immunisation by i/v Ig can be done.

◆ Others –

- Cyclosporine
- Nifedipine
- Pentoxifylline
- Progesteron (in doses of 10^{-5} mmol/L can inhibit in vitro TH1 immunity to trophoblast.)

10. **Environmental / Occupational factors** –
    No specific treatment except counselling.

Prognosis for viable birth

Following

One abortion	-	76%
Two abortion	-	70%
Three abortion	-	65%
Four abortion	-	60%

With

Genetic factor	-	20-80%
Anatomic factors	-	60-90%
Endocrine factors	-	>90%
Infectious factors	-	70-90%
APA syndrome	-	70-90%
TH1 cellular immunity	-	70-87%
Unknown factors	-	40-90%

Following

USG detected fetal cardiac activity at 6 wks of gestation – 77%

References:

1. Current obstetrics and Gynaecology vol. 9 Issue 1 March 1999. Topic - Current concepts in pathogenesis of Recurrent Miscarrage (P 2-6) M. Thlppala, D. Ylikorkala
2. Antiphospholipid antibodies and investigation of recurrent Abortions M.J. Hewitt, S.M. Quenby, P,.N. Baker
3. The Medical and Surgical Management of Recurrent Miscarriage Pg. 13 – 18. J. Walker
4. Psychological aspects of spontaneous and Recurrent Abortions (P 19-22) D. Bagchi, T. Friedman
5. Gestational Diabetes (P 23 – 28) F.D John Store.

Event Highlights

medicare 2004 INDIA news

6-8 April 2004 | Pragati Maidan | New Delhi | India www.medicare-expo.com

The Market :

- Health Market worth Rs. 10,00,000 million
- Market Expected to reach Rs. 20,00,000 million by next three years
- Growing at 17 % per year
- 70% of hospitals are private
- Population growing at 2.5%
- Pharmaceutical market is worth Rs. 240,000 million
- Dental Market is worth Rs.1,00,000 million
- Specialist Dental equipment Industry is worth Rs.667 million
- Biotechnology market is worth Rs. 95, 000 million
- General surgery equipment market is worth Rs. 31, 500 million
- Imaging equipment market is worth Rs. 11, 500 million
- Clinical instruments market is worth Rs. 5,800 million
- Critical care equipment market is worth Rs. 3, 300 million
- Cardiac surgery devices market is worth Rs. 2, 600 million
- Self care equipment market is worth Rs. 3000 million
- Ophthalmology equipment market is worth Rs. 2500 million
- Urology equipment market is worth Rs. 1500 million
- Other medical hardware market is worth Rs. 5300 million
- 15,000 hospitals
- 875,000 hospital beds
- 500, 000 doctors
- 600,000 nurses
- 170 medical college
- 200,000 retails chemist outlets
- 18, 000 new doctors every year

Government commitments: Healthcare investment by 2010

- Increase government health expenditure to 2% of GDP and total health expenditure to 6% of GDP
- Increase central government health grants from 15 to 25% of total health spending
- Increase utilisation of public health facilities from less than 20% to more than 75%
- Increase government-funded health research to 2% of total health spending

The Show

- First major international healthcare event in India with special focus on Dentistry, Laboratory & Healthcare
- Exhibition showcasing the latest cutting edge technology from over 25 countries
- Dedicated Dentistry, Laboratory & Healthcare seminars and hands-on workshops
- Round table 'Meet the Buyers" sessions.
- International and national visitors and delegates.
- Foreign national pavilions

The Benefits

- Meet with specialists across the globe.
- Huge opportunities for collaborations and tie-ups.
- Meet with decision makers from India and overseas private and public sector
- Keep up- to -date with the latest development, products and techniques in dentistry, laboratory and healthcare services
- Show case your products and services to the potential audience

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- Nursing Homes
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- Orthopedic Equipment and Services
- Pharmaceutical Distributors
- Pharmaceuticals Manufacturers
- Physiotherapy Equipment and Services
- Private Clinics
- Specialty Hospitals
- Telemedicine Equipment's
- Telemedicine Services
- Waste Management Providers

.........and many more

Visitor's Profile

- CEO's, Technocrats and Scientists
- Marketing Chiefs, Doctors and Consultants
- Manufacturers of Equipment Suppliers, Entrepreneurs.
- Policy Makers, Diplomats and Foreign Commercial Missions
- Medical Associations and Trade Delegations from India and overseas
- Buying Missions from neighboring regions.
- Key Government Officials, Government bodies representatives, Dr. students from Medical, Dental Colleges/ Institutions, Academicans

Publicity Plan

- Integrated publicity camping for promotion
- Target market publicity through Medical, Laboratory & Dental Journals, Magazines, Websites, Direct Mailers, Print & Electronic Media.
- Media persons from Health Magazines, Journalists, Press Reporters, TV Crew, etc.
- Through International, National & Regional Conferences & Exhibitions.
- Through Indian & Foreign Missions and Overseas Office and Marketing Associates.

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- New Product Launch
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For details contact: Rajeev Chadha at C-60, Nizamuddin East, New Delhi-110013, Tel :+91 11 24352141/44, Fax : +91 11 24354077/5215, Email : medicare@tafcon.com

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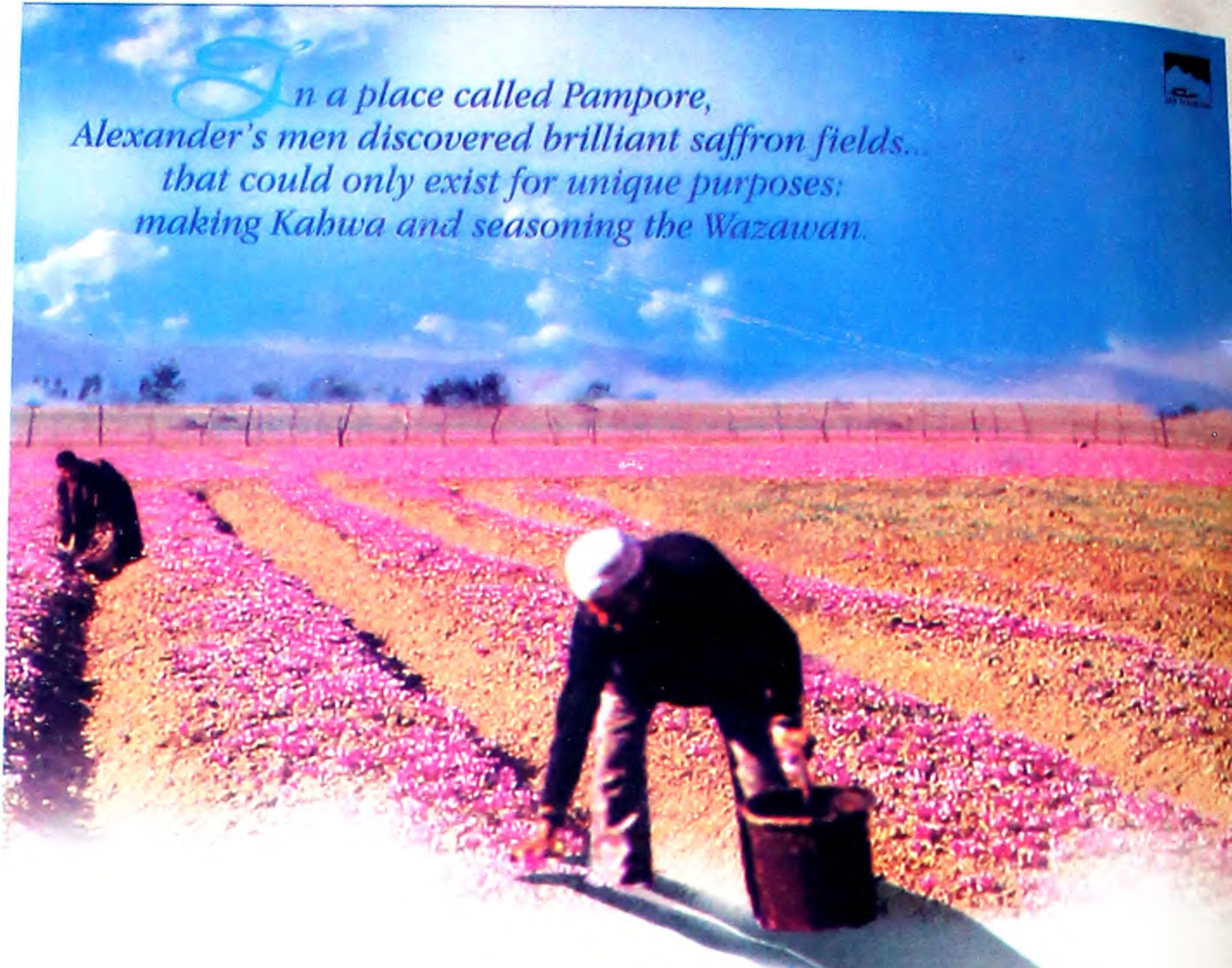
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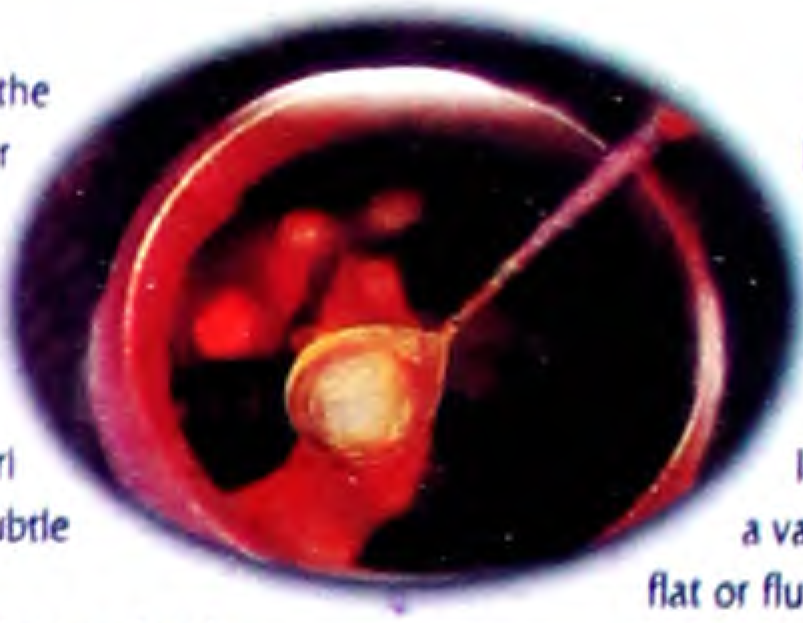

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